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ALLERGIC DISEASES OF THE EAR.*†

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Duke[1] reports two cases of Ménière's syndrome. In each case there was decreased hearing, but the report does not show that the present attack had anything to do with the hearing defect. Each patient was a marked case of allergy. In each case the Ménière complex was relieved by epinephrine. In each case the Ménière complex was partly or wholly relieved by the avoidance of foods to which the patients gave positive intracutaneous tests. In each case the Ménière's syndrome was reproduced by the administration of these foods. Kobrak[2] discusses the causes of crises of the VIIIth nerve. He considers allergy as one of the causes of this condition. His discussion is rather a theoretical one but is illustrated by cases. The crises of the VIIIth nerve are to be explained by changes in the VIIIth nerve or by disturbances in surrounding tissues affecting it. Several years before this communication he advocated the explanation that VIIIth nerve crises may be due to angioneurotic edema and that in VIIIth nerve crises allergy should be looked for. He was led to this conclusion by two observations: First, in three cases owing to a false diagnosis the posterior fossa was entered, and when the dura was uncovered, there was a discharge

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of great quantities of fluid. Second, in some cases of crises of the VIIIth nerve he observed elevation of pressure with lumbar puncture. He thinks that in crises accompanying not only angioneurotic edema, but migraine and epilepsy, allergy should be sought for. He stresses the importance of a good history. He mentions a patient who developed an VIIIth nerve crisis for the first time when he visited a goose farm. A second patient had hay fever for three years and developed with the hay fever a middle ear inflammation accompanied by vertigo which lasted for several months and then disappeared in a very violent watery head cold. Dederding[3] reports three cases of angioneurotic edema. In addition to the involvement of the skin and mucous membrane, there was bilateral tinnitus in the ears, poor hearing and attacks of severe vertigo. The vertiginous attacks were accompanied by a rotary nystagmus which lasted for several hours. In some attacks the hearing was only diminished in one ear, particularly during the later attacks. The hearing returned to normal with the disappearance of the edema. The landmarks on the drum were sometimes less marked during the attacks. Proetz[4] observed in 1931 that the allergic reactions in the labyrinth are essentially those of sudden vestibular irritations with inconstant cochlear disturbances. He states that the patient is seized with attacks of vertigo, usually accompanied by some nystagmus accentuated upon looking to the right or left. There is often a high-pitched tinnitus and slight loss of hearing on the affected side. In the intervals between the attacks the vestibular reactions are normal and the hearing may be unimpaired. He describes a male, age 48 years, who complains of intermittent tinnitus and vertigo for the preceding five years. Two exceptionally severe attacks had occurred two years and four weeks, respectively, before the first examination. The first severe attack was preceded by increased deafness in the right ear, associated with nausea and vomiting. During an attack rotary nystagmus was noticed on looking to the right. Caloric and turning tests caused violent discomfort. Both the caloric and turning tests gave negative results. The allergist found the patient sensitive to milk, butter and cheese. Three subsequent attacks followed the ingestion of butter. Eating foreign protein to which the patient was sensitive caused an edema of the tympanum which is transient in character.

There was some reduction of hearing due to the mechanical damping of the cochlear apparatus. The drum was red and swollen. Patient complained of intense pain, roaring and partial deafness in one ear. Myringotomy produced a drop of serum and the incision healed promptly. Dr. Proetz has furnished us with the subsequent history of this case, which will be referred to later. Portmann[5] describes vasomotor disturbances of the inner ear but does not consider allergy as an etiological factor. Duke[6] describes the general symptoms for shock or severe allergic reactions in hypersensitive people. Amongst other things, he mentions deafness and Méniére's syndrome. He states that in two cases patients suffered from tinnitus, nausea and vomiting, and fell helpless to the ground. In one case the vestibular reactions were subnormal. Hearing was decreased one-fifth for both bone-conduction and high tones. In the second case there was reduced hearing with increased bone-conduction on the right side and subnormal labyrinthine responses. No observation was made regarding the hearing of the patient before or after the attack. Yandell[7] states that he has definitely established orris allergy as an etiological factor for the Méniére's syndrome. Yandell's case suffered from periodic attacks of vertigo, tinnitus, nausea and vomiting for 14 years. No mention is made of deafness. The administration of a very small dose of orris produced the attack which lasted six hours. After tolerance to orris was established, these attacks disappeared. The author expresses the opinion that the attacks were due to an edema of the labyrinth. Vaughn and Hawke[8] report, amongst other unusual manifestations of angioneurotic edema, Méniére's disease, nystagmus, meningismus; generalized cephalic disturbances generally accompanied the peripheral nerve manifestations and all probably due to an attack of angioneurotic edema in the brain substance. He considers that allergy should be considered as a possible etiological factor in these conditions and that the absence of an allergic family history does not rule out allergy. Vogel[9] reported that of 83 patients with Méniére's complex, five had hay fever, nine urticaria, 15 nonallergic vasomotor rhinitis, five asthma. Urbach and Wilder[10] report a case of Méniére's symptom complex which went on to unconsciousness with urticaria. This picture appeared following an injection of horse serum. He states that while the Méniére symp-

tom complex is not often allergic, we must consider allergy as one of the causes. Bogaert[11] states that cerebral urticaria produces unilateral hemiplegia, convulsions, unilateral hemianopsia, somnolence, and symptoms of cerebellopontine angle lesions. Furstenberg, Lashmet, and Lathrop[12] reported the results of the treatment of Méniére's symptom complex by decreasing the intake of sodium and the prevention of the accumulation of sodium by the body. Wasowski[13] reports that the hypodermic administration of adrenalin or the application of adrenalin to the mucous membrane of the promontory has a favorable action on tinnitus and vertigo when the cause is in the labyrinth. He experimented on cats to determine the action of adrenalin on the normal labyrinth when injected intravenously. With intravenous injections there were slow, wide movements of the eyeball. If the adrenalin was injected into the tympanic cavity and the injection repeated several times, the caloric reaction disappeared. Malone, cited by Bray,[14] reported two cases of Méniére's disease which responded to desensitization for orris root. Rowe[15] reviews Duke's two cases with the Méniére's syndrome and adds one case of his own. No mention is made of the patient's hearing during the attack. Rowe and Richet[16] mention a patient who suffered from Méniére's disease due to allergy. In discussing the symptoms of their sensitized animals, they do not mention vertigo as ever having occurred. Lewis[17] describes six cases of acute otitis media which he thought were due to allergy. Craft[18] states that eczema of the external ear and migraine may be due to allergy. Watkins[19] reveiws the subject of allergy in otolaryngological factors. He does not mention any ear conditions as being due to allergy. Levy[20] discusses tinnitus due to allergy. He reports a patient who suffered from tinnitus from the age of 16 until 49 years, when it became almost unbearable. This was a female. The tinnitus appeared at the age of 16 years with urticaria. He is of the opinion that allergic tinnitus may appear with or without the Méniére symptoms. He did not prove that in his case the tinnitus was definitely due to the allergy. Bray[21] cites heterogeneous groups of metals causing allergic reactions. The mucous membrane is rarely affected in drug allergy. The chemical probably combines with the body protein and the combination acts as an antigen and produces the allergic symptoms. Hansel[22] has the most complete review to date

of allergic lesions of the ear. He speaks of allergic eczema of the auditory canal, of contact dermatitis in the external ear, the result of hypersensitivity to drugs. He refers to the paper of Lewis in 1929 and of Proetz in 1931, regarding allergic otitis media. He winds up with the statement which I agree with in every way: "In allergic individuals it is quite impossible to make a diagnosis of allergic otitis media unless a marked predominance of eosinophils is demonstrated in the aural discharge."

He discusses at length the Méniére symptom complex. He calls attention to the fact that this symptom complex is characterized by vertigo, deafness and tinnitus. With our cases of intense vertigo, in two out of three there was no history of deafness and neither were we able to obtain any evidence of loss of hearing. He has made a careful review of all cases of the allergic Méniére's syndrome to date. Will not review the extensive bibliography.

During this investigation all patients with allergic rhinitis were questioned as to ear symptoms. Almost all of the patients studied were cases of perennial allergic rhinitis. This is usually a mild form of allergy and yields nicely to treatment. Approximately 20 per cent of the cases of allergic rhinitis that have been studied presented ear symptoms. Some patients presented themselves with the ear symptoms as the outstanding complaint. This was particularly true of the Méniére symptom complex. As suggested by Duke many years ago, this complex develops in individuals who are so allergic as to develop shock when exposed to the allergen to which they are sensitive. All our patients with the Méniére symptom suffered from a very severe type of allergy.

Allergic patients presented an interesting series of symptoms. Table I is a list of these symptoms in the order of their frequency.

These symptoms made their appearance during spontaneous allergic attacks or when allergic tests were being made. With the appearance of the symptoms occasionally there would be changes in the drumhead. The luster would change and there was a slight injection of vessels along the handle of the malleus. These symptoms, when not due to tests, were usually controlled satisfactorily by allergic treatment with the exception of tinnitus, which will be discussed later. The

symptoms were never produced by skin tests. They were caused by mucosal tests, leukopenic index tests and by contact with or the feeding of allergens to which the patient was sensitive. With the leukopenic index test, symptoms usually appeared in about 20 minutes and lasted for several hours; with the intramucosal test, symptoms appeared much sooner.

TABLE I.

Stuffiness or fullness of ear or ears.

Subjective loss of hearing.

Shooting or dull pains in ears.

Itching or sensation of something crawling deep in ear.

Continuous high pitched tinnitus.

Vertigo.

Nausea (especially when ear reactions are precipitated by a leukopenic index test but also independent of this)

Extreme nervousness and irritability.

Hyperacusis.

Syncope.

TABLE II.

Itching of the ear.

Fullness in the ear.

Pounding in ears.

Ears feel blocked.

Earache.

Feeling of tightness and of drawing about the ear.

The ear feels swollen.

Feeling of pressure in the ear

Nausea and dizziness.

Decreased hearing.

Table II is a list of the subjective ear symptoms appearing with positive findings when the leukopenic index tests were made. The description of the symptoms is given in the patient's own words.

Two cases of subjective hyperacusis were studied. They were both very sensitive to high tones. One, D. H., would not tolerate high notes on the audiometer. Her attacks came

on spontaneously and were also caused by intramucosal tests, using substances to which she was sensitive. She secured subjective relief by the use of adrenalin.

With the use of adrenalin her hearing improved as measured with the audiometer (see Fig. 3). With allergic treatment the hyperacusis disappeared. The second patient, R. M., also had spontaneous attacks of hyperacusis, and it made its appearance with the intramucosal tests for oats, wheat and potatoes, to which he was sensitive. Occasionally he became irrational when tested with these substances. Spontaneous hyperacusis disappeared as long as he remained on his diet. Unfortunately, he was on relief and had great

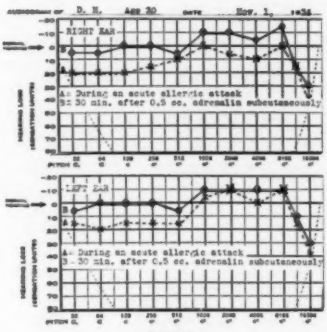


FIGURE 3.

difficulty in controlling this, and when he deviated from the prescribed diet, he had recurrence of symptoms.

There was no relationship between the severity of the symptoms and the changes in the drumhead. Ear symptoms were usually controlled by the use of adrenalin in ten to 30 minutes with the exception of tinnitus. The tinnitus would be improved. If the tinnitus was produced by the mucosal test, it would disappear in ten to 20 minutes after the administration of adrenalin. One patient, when fed on wheat for one week in order to prepare her for the leukopenic test, developed one hour after the eating of wheat on every occasion a subjective sensation of the ears being stopped up, marked tinnitus, and slight vertigo. When, as in this case.

tinnitus appeared only during the allergic attack, it could be controlled by allergic treatment. One patient with tinnitus all of the time and exacerbations characterized by itching, blocking of the ears, subjective decrease in hearing which could not be confirmed by the audiometer test, had the tinnitus and all ear symptoms disappear with allergic treatment.

Almost all of our patients that complained of loss of hearing had a decrease that could be measured with the audiometer. Occasionally a patient would have subjective loss of hearing which could not be confirmed with the audiometer. Some of our patients who were allergic and suffered from tinnitus had the tinnitus due to something other than allergy. All of these patients that did not improve with allergic treatment had evidence of chronic middle ear changes. One such patient had the tinnitus made much better by allergic treatment.

Most marked changes in the audiogram were in the lower part of the tonal range. In some cases the low notes only were affected. In others the hearing for all notes was disturbed in about the same degree. In only one case was there a suspicion that the high notes were affected more than the low.

Characteristic of deafness due to allergy is a variation from day to day, from week to week, and from year to year without objective changes in the tympanic membrane. The same variation is noted in the function of the static labyrinth. With the caloric test using water as cold as 48° Fahrenheit, one may get no reaction, but after the patient remained free from attacks of vertigo for one year, get a normal reaction with water at 68°. (T. C.) One may have poor hearing as demonstrated by the audiometer two months after the last allergic attack and then a few months later find the hearing perfect. (V. C.)

The itching which the allergic patients complain of is always deep, never superficial, and may be accompanied by itching in the roof of the mouth. The pain may be over the mastoid region or it may be like the earache of childhood, sharp and shooting in the ear.

During the allergic attacks one may recognize in different patients different groups of symptoms which constitute a syn-

drome. We have divided our patients into groups, dependent upon the syndrome which they present.

The first condition is one that is apparently a crisis of the VIIIth nerve. Many writers refer to it as the Ménière symptom complex; however, all patients do not have a loss of hearing that can be detected.

Group 1: In our service at the Institute we have had two outstanding cases of the labyrinthine type of vertigo due to allergy, and Dr. Proetz has permitted us to include a case that he has observed since 1924. His report, published in 1931, appears in the bibliography.

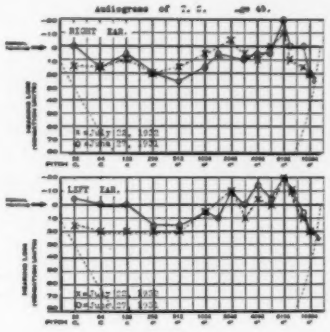


FIGURE 4.

T. C., male, age 45 years, suffered from migraine. Six weeks before his present attack he awoke in bed at night very dizzy. He was afraid that he would fall out of bed. He was seen by two physicians, who reported no nystagmus, no tinnitus, and who said that his hearing seemed to be quite all right. In four days he was able to get around, but was very unsteady on his feet. Stooping down or turning his head would cause vertigo. The day preceding his attack he had eaten something which disagreed with him. He was allergic to certain pollens. The general examination was negative. There was nothing suggestive of infection about the nose, throat or teeth. There was no spontaneous nystagmus. Caloric right: Using water at 68° F., no reaction in three and one-half minutes. This was repeated with water at 48° F., with no reaction.

Caloric left: Using water at 68° F., nystagmus appeared in 25 seconds. Marked vertigo appeared before the nystagmus. His hearing as determined with the audiometer was practically normal. With the avoidance of certain foods to which he was sensitive, his attacks disappeared except for some slight attacks of vertigo which came on after an indiscretion of diet. He was examined one year later. The hearing was practically the same. With water at 68° F., right ear now reacted in one minute and 30 seconds, and the left ear in one minute and 30 seconds. This patient has had no return of his trouble.

The second case, M. A., female, age 30 years, developed intense labyrinthine vertigo which made its appearance with a sudden development of allergic asthma. The hearing was nor-

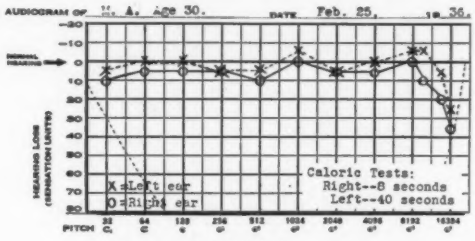
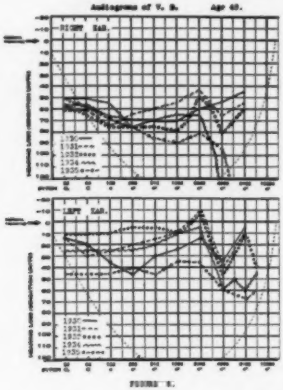


FIGURE 5.

mal. Made a day after an acute attack of vertigo, caloric test with water at 68° F., reacted in eight seconds on the right and 40 seconds on the left. During the attacks of vertigo there was a rotary nystagmus to the right and on some occasions to the left. At the height of her dizziness it was present to the right and to the left, and she presented a definite vertical nystagmus. Allergic treatment did not relieve her of her attacks of vertigo. She was only relieved by the use of the Furstenberg diet.

The third case was Dr. Proetz's patient. V. B., male, age 49 years, developed the Méniére symptom complex with severe high-pitched tinnitus. The attacks were so severe that the patient was incapacitated. Between the periods of attack the hearing was normal and vestibular tests gave negative results. This was in 1924 and 1925. When seen during an attack

patient had rotary nystagmus to the right on looking to the right, horizontal nystagmus to the left on looking to the left. The patient was sensitive to milk and butter, and received complete relief except on two occasions when butter was eaten. This patient has been under observation up until the present time. In 1933 the patient noticed that he was very deaf during an intestinal upset. As is often true with allergic patients, he developed sensitivity to other foods and to iodides and later on to pork. The preceding chart shows the changes in his hearing for right and left ears from 1930 up to date. The caloric test was repeated in 1935; the right ear did not



react in three minutes and 30 seconds; the left reacted in one minute and 30 seconds.

It is interesting to note that particularly in the left ear the patient heard better in '31, '32 and '34 than he did in '30. The hearing in both ears was the poorest in '35. Throughout the year the patient has been under observation, he has had occasional attacks of vertigo. I trust that in his discussion Dr. Proetz will give us a report on the condition of this patient at this time.

It is interesting to note that occasionally a sensitized rabbit shows the same symptoms as an animal that has had the semicircular canals traumatized. We noted a rabbit that had

been sensitized to egg white. It turned constantly to the right. When walking around a room, it propped its right side against the wall in order to make progress in a straight line. There was no evidence of nystagmus. The ear canals were clean. When held by his hind legs, he very definitely turned his head to the right, keeping it in the plane of the long axis of the body. In the cage he propped his right side against the wall.

Group 2: Méniére's symptom complex with unconsciousness. J. Y., male, age 49 years, since age 22 years, whenever he has accidentally eaten crisco he has developed an itching in the auditory canals, tinnitus aurium, and vertigo so severe that he has fallen to the floor. He develops an urticarial

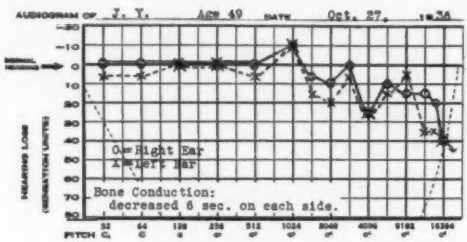


FIGURE 7.

edema of the lung and glottis so severe that at times a tracheotomy has been considered. No nystagmus has been reported. When he has an attack, physicians are immediately sent for. His hearing gets poor. He notices that he can see people's lips move, but he cannot hear any sounds and then he loses consciousness. He has had many attacks and the audiograms taken between the attacks are almost normal. One would feel that in this case there was an edema of the brain with or without involvement of the labyrinth. I have been unable to find a report of a similar case except when the attack was due to the injection of horse serum.

Group 3: This is by far the larger group of cases. They are characterized by decrease in hearing with positive Rinné. With a decrease in hearing there is a sensation of fullness in the ear, usually itching, earache, tinnitus, and, not so com-

mon, vertigo or nausea, or both. One patient had a pulsating tinnitus; all the other tinnitus was constant. This syndrome occurred during a spontaneous allergic attack, when intramucosal tests were made, and when a leukopenic test was being performed. The following two cases will serve as examples. Almost all patients were relieved of these symptoms by allergic treatment with the exception of tinnitus as noted earlier in the paper. All these patients had their symptoms controlled by the use of adrenalin. The prognosis of this class of cases is good because, as we said before, they are associated with perennial allergic rhinitis which, as compared with other allergic things, yields readily to treatment.

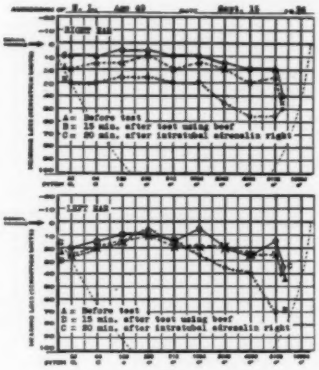


FIGURE 6.

The following case is an illustration of this group: N. L., female, age 48 years, entered the clinic complaining of attacks of nasal blockage, headaches, dyspnea, sneezing, pain in the ear, fullness in the ear, slight deafness, and slight deterioration of vision, all occurring during an allergic attack. The patient suffered from the perennial type of allergic rhinitis. The blood eosinophilia was 2 per cent. There was an increase of the eosinophils in the nasal smear. The patient was particularly sensitive to wheat. Her skin test to wheat was 2+; it gave a marked mucosal reaction, and with the leukopenic index test there was a drop of 2,325 cells in 60 minutes. With her intramucosal test and leukopenic index test she developed the same symptoms as during spontaneous

allergic attacks. During the week preceding her leukopenic index test she was fed wheat to prepare her for the test. The eating of the wheat produced in a mild degree the symptoms for which she came to the clinic. She also was sensitive to beef and rye. This patient presented so many interesting symptoms that when she was tested for beef, audiograms were taken before and after the test. This is the one patient whose audiogram showed a slightly more marked decrease in the hearing of high notes than the low notes. Using beef, a mucosal test was made. In the right ear there was a decrease in the power of hearing all notes. This was a little more marked with the high notes. In the left ear the decrease in

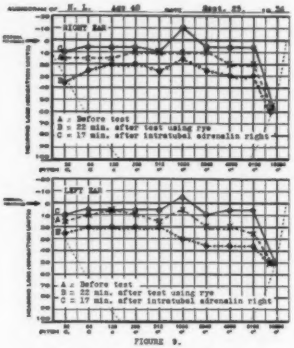


FIGURE 9.

hearing was for notes above 512. With the use of adrenalin intratubally on the right side, the hearing, right, became slightly better than before the test was made and on the left it approximated the audiogram made before the test. In all our patients the same reaction was secured, whether the adrenalin was placed in the Eustachian tube or given hypodermically.

This patient said that when the adrenalin was given, her vision improved. Consequently, when the mucosal test for rye was given, an ophthalmologist was present and tested the vision at the same time as the hearing was tested.

With the intramucosal test there was a decrease in the hearing in each ear. In this instance the left ear was affected

the same as the right. The hearing for high notes again in the left ear was decreased a little bit more than the low. After the administration of rye there was a marked decrease in vision. With the intratubal administration of adrenalin, right, the hearing and the vision improved so that both were better than before the test.

A second patient in this group, F. S., male, age 25 years, had at all times a loss of power to hear high notes, as shown by the audiogram, which did not vary with the allergic attack or with the administration of allergens. The audiogram taken between the allergic attacks showed normal hearing up to 1,024; then there was a sharp drop (see audiogram F. S.—A).

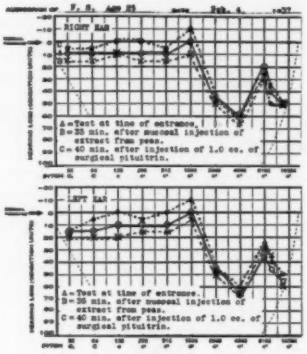


FIGURE 10.

This patient gave ear symptoms with almost all of the intramucosal tests which showed a positive reaction. He gave positive leukopenic index test with seven allergens. Four of these produced ear reactions. Fig. 10 shows the audiogram before an intramucosal test using peas, 35 minutes after the mucosal test, and after the administration of surgical pituitrin subcutaneously. This was given immediately after taking the second audiogram. The change in the audiogram with the administration of the peas and the use of the pituitrin was confined to the low notes. The high notes were not changed.

This patient was observed over a period of months. His ability to hear low notes was decreased by the administration of allergens. The hearing for high notes remained always

the same, as demonstrated with the audiometer. The high tone loss was probably due to something other than allergy. As a result of allergic treatment his rhinitis was controlled and subjectively he heard better. After treatment the defect in the high notes was the same as before.

Dr. Bunch reports a case sent to him with a diagnosis of allergic deafness. There was a loss of power in hearing high notes. The patient was a naval officer and Dr. Bunch considered his high note loss due to exposure to very loud noises.

Patients with nonallergic defects have not had the audiograms changed by the use of adrenalin or surgical pituitrin.

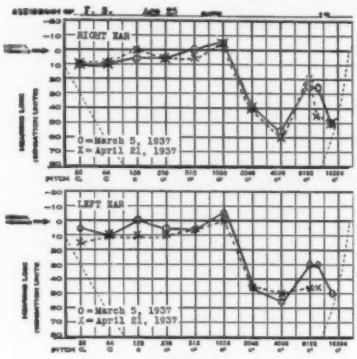


FIGURE 11.

There is a fourth group of cases characterized by migraine and ear symptoms. Perhaps one of the cases in the Ménière group, T. C., should be placed in this class. These patients, like those suffering from Ménière's syndrome, are very allergic.

One patient, V. C., female, age 21 years, illustrates this group. She suffered from scintillating scotomata and migraine since the age of 19 years. She was incapacitated and unable to do housework. She had been studied in the ophthalmological and medical services. She was referred to our service for examination. Nothing was found about the nose or throat to account for her trouble. We felt quite positive, however, that

from a complete allergic study she would receive benefit. With her attacks she developed scintillating scotomata, followed in an hour by excruciating vertex headaches. At this time there was a feeling of stuffiness in the ears, loud tinnitus, nausea, vomiting, diplopia, poor vision, and nystagmus to the right on looking to the right. The nystagmus was present only when the vertigo was marked and disappeared with the vertigo.

The subcutaneous injection of adrenalin always controlled these attacks. During the attack the patient was too ill to have caloric tests made. Such tests made in the intervals

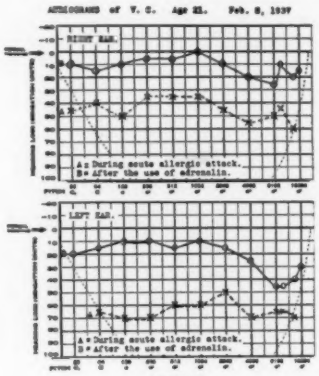


FIGURE 12.

between the attacks showed nothing of significance. Fig. 12 shows audiograms taken during the acute attack and after the use of adrenalin. There was no increase in eosinophils, either in the nasal discharge or in the blood. In addition to other things, she was sensitive to whole wheat. When the leukopenic test was made for wheat, she developed a headache, nausea, had blocked ears and tinnitus.

It was decided with this patient, if possible, to get an audiogram preceding the attack, during the attack, and after the injection of adrenalin. Fig. 13 shows an audiogram taken the evening before an acute allergic attack, one taken at the time of the scintillating scotomata, then adrenalin was given

subcutaneously and an audiogram taken five minutes after the adrenalin and 40 minutes after the administration of adrenalin. Note how the hearing decreased in each ear and that 40 minutes after the administration of adrenalin the hearing was as good as the day preceding the attack. This patient was placed on an allergic diet with positive nutrition. On April 28, 1937, she had been free of the severe allergic upsets with scintillating scotomata and headache for four months. During this time she had some mild attacks characterized by a feeling of fullness in the ears, itching, vertigo, and subjectvie deafness. Her hearing was determined with

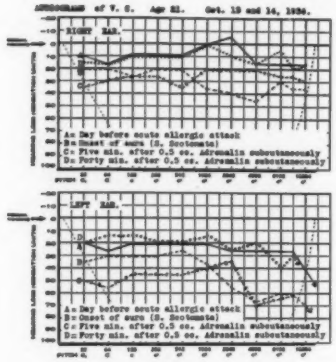


FIGURE 13.

the audiometer at this time, and the findings in both the right and left ear were quite normal. The only residual trouble with her ears is a slight tinnitus. In March she had a slight rhinitis lasting for five or six days, the etiology of which is questionable. It was found that her attacks were made worse by the administration of codein. We suspected that she might be sensitive to the codein and giving it would make the attacks worse instead of better.

On March 22, 1937, when she was free of all symptoms, the hearing in each ear was tested and 0.3 gm. of codein was administered. The patient developed headache, nausea, vomiting and tinnitus. Fig. 14 showed the loss of hearing following the administration of codein and the improvement in

hearing following the administration of adrenalin subcutaneously. It is interesting to note that the hearing in the left ear was markedly decreased before the administration of the codein. The audiogram, right, approximated the original, while in the left ear it showed an improvement of some 30 to 40 sensation units over the original. On April 28, five weeks later, the hearing in the left ear was quite perfect. The leukopenic index test for codein was negative. This is to be expected, as we could not prepare the patient for her test by administering codein, as we give food during the preceding week. The skin test using codein gave a marked positive reaction. This is interesting because drugs to which indi-

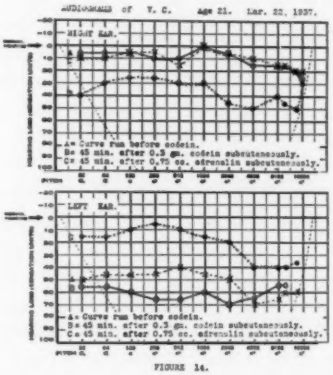


FIGURE 14.

viduals are sensitive do not usually give positive skin reactions.

Otitis media is a common complication of allergic rhinitis. The common finding in allergic rhinitis is a very edematous pharyngeal tubal cushion. It is only reasonable to suspect that there will be a blockage of the tube with resulting changes in the tympanic cavity. In our experience, we have found more cases of acute otitis complicating allergic rhinitis during inclement weather. That is the period when we have acute infectious otitis media. It is interesting to note that during the two-year period we have made a study of allergic rhinitis, acute otitis media has been a very unusual complication of the cases studied. The patients with acute otitis

media differ from those in our third group with hearing defects, in that the patients mentioned as having allergic deafness, with one exception, always had a positive Rinné. Hansel[23] has an excellent review of otitis media. He refers to Lewis'[24] and Proetz's[25] contribution to our knowledge of this subject. We agree with Dr. Hansel's observation that it is quite impossible to make a diagnosis of allergic otitis media without finding an increase of eosinophils in the ear discharge. We have had one case of chronic otorrhea in a woman, age about 35 years, where the ear continued to discharge after nine months of conservative treatment. This patient had an allergic rhinitis with a marked chronic vasomotor rhinitis. With the control of the allergic rhinitis and eradication of the chronic vasomotor rhinitis, the ear ceased to discharge and has remained dry for more than a year. Five years ago I became interested in acute otitis media in allergic individuals. During this time members of the staff who have been interested in this phase of the subject with me often said, "Now we have a typical case of allergic otitis media." In each instance the acute ear has developed during an acute upper respiratory upset. They were all typical cases of mild acute otitis media with slightly reddened or distinct inflamed bulging drumheads. When it was necessary to incise the drumhead, an increase in eosinophils in the discharge was never found. We have only had two cases of purulent otitis where there was an increase in the eosinophils in the ear discharge. These were both cases of chronic otorrhea. There was nothing in the history or examination suggestive of allergy, and in the material removed at operation there was no suggestion of allergy. Following operation the ears healed nicely. The eosinophilia was undoubtedly due to the chronic infection. It is interesting to note that while we do not have an increase in the eosinophils in the ear discharge of cases of acute otitis media complicating allergic rhinitis, we have found an increase in eosinophils in material taken from the tube 1 cm. from the pharyngeal tubal orifice. So we think we may assume that the pharyngeal end of the tube at least enters into the allergic process. We have not proven that the tympanic end of the tube and the tympanum itself are not involved in it. These patients do not come to operation or autopsy. Consequently we have not been able to study the pathology. Certainly repeated attacks of acute

otitis media in allergic individuals will have the same deleterious result on the hearing as occurs in repeated mild attacks of acute otitis due to adenoids.

The cases under consideration are treated the same as any other mild infection of the ear with a good result so far as the individual attack is concerned. Acute allergic sinusitis is characterized by an absence of redness in the nasal membrane, and it will not yield to treatment as usually given for an acute infection. The acute otitis with allergic rhinitis is accompanied by evidence of inflammation and does yield nicely to treatment usually given for mild acute infections. The acute allergic sinusitis is a different clinical entity from the acute otitis which accompanies allergic rhinitis. It yields only to allergic treatment.

To prevent the repeated attacks of acute otitis complicating allergic rhinitis, it is necessary to control the allergic rhinitis and eradicate the chronic vasomotor rhinitis which results from it.

Dermatitis of the external canal and ear is often of allergic origin and will yield only to allergic treatment. In the same way with skin lesions in other parts of the body, food allergy, bacterial allergy and drug sensitivities must be given consideration if the best result is to be secured.

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INDUCED NYSTAGMUS IN MONKEYS FOLLOWING PERIPHERAL VESTIBULAR LESIONS (WITH CLINICAL CORRELATIONS).*†

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In a previous report[1] we related some observations on induced nystagmus in monkeys, along with the general effects on the animals following unilateral and bilateral labyrinthectomy and intracranial section of the VIIIth nerve. Studies of the effects of similar vestibular lesions in monkeys have been continued since that time and some observations that seemed to us of clinical importance here are reported.

Although extensive studies of the vestibular system in experimental animals have been made by physiologists, psychologists and physicians since Flourens[2] discovered the vestibular end-organ over 100 years ago, the information concerning the vestibular function so obtained has only to a small extent served any useful purpose in the care of the sick. The value of such fact-finding procedure in research is proportionate to the extent of our being able to correlate the findings with the symptoms appearing in patients with disturbances of vestibular function. Although there is no general agreement on all of the effects of peripheral vestibular lesions, either in experimental animals or in man, our observations on the phases of the problem reported here are in general conformity with those of other investigators on lower forms of mammals and those of Uffenorde[3] and Magnus[4] in the monkey. The results of comparing our observations on monkeys with those in patients having unilateral vestibular lesions enable us to have more than a speculative opinion on a few of the reactions of the rotation, caloric and galvanic tests.

Experimental Procedures: Macacus rhesus monkeys averaging eight to 10 pounds were used. In all, over 20 monkeys

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have been used to date. Rotation, caloric and galvanic tests for nystagmus were performed in all cases before any operative procedure was carried out. Rotation was performed in the box described by Northington and Barrera.[5] The post-rotatory nystagmus time in normal monkeys averaged 15 seconds following rotating 20 times in 20 seconds, eyes not shaded. The caloric testing was done by the running of water at 40° F. slowly into the ear canal. Five cc. usually sufficed to produce a vigorous response. The galvanic test was carried out with the simple clinical galvanic testing battery. Leads carried from the two terminal poles of the circuit formed the stimulating electrodes, the polarity of which could be varied at will by a pole-changing switch. Contact with the ears was made by covering the tip of the electrode with cotton, moistening this with normal saline solution and then inserting the tip into the external auditory canal. In the average monkey with the electrodes in the two ears, a good typical response could be obtained at current strengths of from 1 to 4 m.a. Testing of the single ears was done by leaving one electrode as usual in the ear tested and applying the other electrode to the back by means of a large, flat, moistened electrode of approximately 8 cm. diameter. With the cathode in the ear and the anode on the back, a good, active nystagmus in the average normal monkey was obtained at about 3 or 4 m.a. of current. With the polarity reversed the response is obtained with about 5 to 7 m.a. Table I is a chart summarizing the responses obtained in the average normal monkey with the three methods of investigation.

In general, two types of operative procedure were used: *1.* Labyrinthectomy, and *2.* intracranial section of the VIIIth nerve. Labyrinthine lesions were performed by a mastoid approach. The technique for labyrinthectomy was essentially that of the Hinsberg operation. Section of the VIIIth nerve intracranially was performed by suboccipital approach to the posterior fossa. Midline splitting of the muscles over the occipital bones well down over the upper vertebrae was performed. The bone was removed well up over the transverse sinuses. The dura and arachnoid were opened and the VIIIth nerve was approached by medial retraction of the lateral lobe of the cerebellum with a small, specially prepared, curved retractor. The trunk of the VIIIth nerve could be seen clearly and it was sectioned usually by a small hook which could be

TABLE I.

NYSTAGMUS REACTION IN NORMAL MONKEY

Tests	Procedure	Head Position	Stimulus	Nystagmus
Rotation	Rotation Right	Face Front	20 Times in	→ 10 to 20 Seconds
Rotation	Rotation Left	Face Front	20 Seconds	← Post-Rotatory
Caloric (40° F.)	Right Ear	Face Down	5 CC.	←
Caloric (40° F.)	Right Ear	Face Front	5 CC.	→
Caloric (40° F.)	Left Ear	Face Down	5 CC.	→
Caloric (40° F.)	Left Ear	Face Front	5 CC.	←
Galvanic	Right Ear +, Left Ear —	Any	2 to 4 M.A.	→
Galvanic	Left Ear +, Right Ear —	Any	2 to 4 M.A.	←
Galvanic	Body +, Right Ear —	Any	3 to 6 M.A.	←
Galvanic	Right Ear +, Body —	Any	4 to 8 M.A.	→
Galvanic	Body +, Left Ear —	Any	3 to 6 M.A.	→
Galvanic	Left Ear +, Body —	Any	4 to 8 M.A.	←

Note: The head of the arrow indicates the direction of the quick component of the nystagmus; that is, ← indicates a horizontal nystagmus to monkey's right.

placed about the nerve and then moved so as to section the nerve. The section usually was made approximately 2 to 3 mm. medial to the interior auditory meatus. In some cases the nerve merely was sectioned and in other cases the stumps of the cut nerve were separated. In most cases the VIIth nerve was cut together with the VIIIth nerve when the lesion was performed intracranially. In these cases ipsilateral complete peripheral VIIth nerve paralysis was present. Very little bleeding usually occurred. If it did occur, it was stopped easily with hot saline. At proper times the various animals were sacrificed and the temporal bones and brains were removed. Serial sections of the temporal bones were stained with the hematoxylin and eosin methods and with Mallory's phosphotungstic-acid-hematoxylin method. The brains were sectioned serially and prepared by either the Weigert or Marchi methods for myelin sheaths.

Observations: The monkeys following nerve lesions, as well as following labyrinthectomy, showed the typical spontaneous symptoms described by us in a previous communication.[6] Following the operation, tests were made at various intervals to determine the responses of the animals to different forms of vestibular stimulation. Three methods of stimulation were used: the rotation, the caloric and the galvanic methods. As previously described, spontaneous nystagmus resulting from unilateral labyrinthectomy or VIIIth nerve section subsides approximately 24 hours after operation. After this time the presence of spontaneous nystagmus did not have to be reckoned with in studying the effects of the induction methods.

Case 1: Table II summarizes the results obtained in the average monkey with unilateral labyrinthectomy in which no degeneration of the nerve resulted.

June 1, 1934: Left labyrinthectomy with usual technique. After operation with the animal well out of anesthesia there was a typical picture of unilateral labyrinthine destruction, including abnormality of motion and abnormalities of posture as previously described by us. Studies of induced nystagmus 48 hours after operation revealed the following: rotation 20 times in 20 seconds to the left gave an average post-rotatory nystagmus time of 14 seconds. This time is the same as that obtained in the monkey before operation. Rotation 20 times in 20 seconds to the right gave an average post-

TABLE II.

NYSTAGMUS REACTION AFTER LEFT LABYRINTHECTOMY (IPSILATERAL VIIIth NERVE NOT DEGENERATED).

Tests	Procedure	Head Position	Stimulus	Nystagmus
Rotation	Rotation Right	Face Front	20 Times in	→ For 7 Secs. } Average for
Rotation	Rotation Left	Face Front	20 Seconds	← For 14 Secs. } One Week
Rotation	Rotation Right	Face Front	20 Times in	→ For 8 Secs. } After
Rotation	Rotation Left	Face Front	20 Seconds	← For 9 Secs. } One Month
Caloric (40° F.)	Right Ear	Face Down	5 CC.	← } Probably More Active
Caloric (40° F.)	Right Ear	Face Front	5 CC.	→ } Than in Normal Monkey
Caloric (40° F.)	Left Ear	Any	50 CC.	No Response Obtained
Galvanic	Right Ear +, Left Ear —	Any	1 to 2 M.A.	→ } Same for All Tests Until
Galvanic	Left Ear +, Right Ear —	Any	2 to 3 M.A.	← } Sacrifice of Animal
Galvanic	Body +, Right Ear —	Any	3 to 4 M.A.	← } Same for All Tests Until
Galvanic	Right Ear +, Body —	Any	5 to 7 M.A.	→ } Sacrifice of Animal
Galvanic	Body +, Left Ear —	Any	3 to 4 M.A.	→
Galvanic	Left Ear +, Body —	Any	5 to 7 M.A.	←

rotatory nystagmus time of seven seconds. On caloric testing, 5 cc. of water at 40° F. injected into the right external auditory canal produced a normal horizontal-rotatory nystagmus. Cold water up to the amount of 50 cc. ran into the left external auditory canal failed to produce any nystagmus. *Galvanic Test:* Ear to ear test: With the cathode in the right ear and the anode in the left ear, nystagmus normal in type was produced at a current of 2 to 3 m.a. With the polarity reversed, making the cathode in the left ear and the anode in the right, a good, active, normal type of nystagmus was produced at 1 to 2 m.a. Ear to back test: With the cathode in the right ear and the anode on the back, good nystagmus reaction was produced at 3 to 4 m.a. With polarity reversed, making cathode on back and anode in ear, good nystagmus reaction was produced at 5 to 7 m.a. The same responses were obtained from the left labyrinthectomized ear. The nystagmus reaction to rotation, caloric and galvanic stimulation were almost identical on the fourth, sixth, tenth and fourteenth day with that of two days after operation. *One Month After Operation:* Rotation tests: Postrotatory nystagmus time on right rotation averaged eight seconds. On left rotation, postrotatory time averaged nine seconds. Caloric testing revealed no nystagmus from the left ear. Normal type of nystagmus was produced from the right ear with 3 to 5 cc. of water at 40° F. Galvanic test revealed normal type, practically equal results from both ears and from either ear to back or back to either ear. Two and three months after operation essentially the same results were obtained as at the one-month test. Animal sacrificed and serial sections of the ears and brain stem prepared. Sections of the brain stem revealed normal appearing VIIIth nerves on both sides. Sections of the ears revealed a partial destruction of the labyrinth on the left and perfectly normal labyrinth on the right. *General Result:* This experiment is typical for all those cases in which a partial labyrinthectomy or puncture of the promontory was performed. They all showed, despite the clinical evidence of vestibular loss on the involved side, a well retained response to the galvanic current stimulation despite the absence of response to caloric stimulation on the involved side. In all these cases no histological evidence of degeneration of the trunk of the VIIIth nerve could be detected. Fig. 1 is a microphotograph of Weigert preparation of the brain

stem of this animal. This demonstrates the normality of both VIIIth nerve trunks, indicating that degeneration had not occurred as the result of labyrinthectomy.

In some cases, however, where the labyrinthectomy had been deeper than in the experiment just reported, the results obtained were different, as shown by the following protocol:

Case 2: Table III is a table summarizing the results of testing in monkeys in which, as a result of unilateral laby-

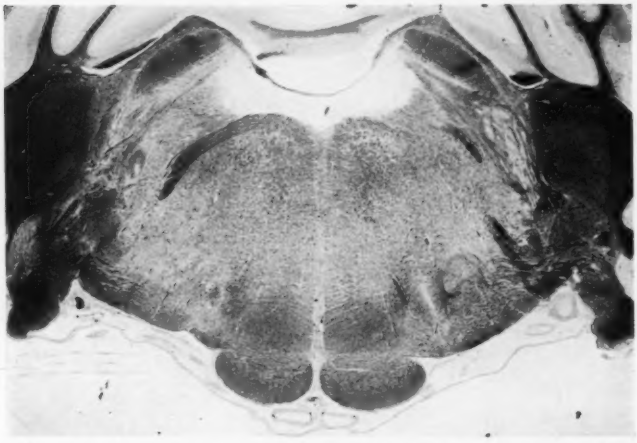

Fig. 1. Frontal section through brain stem of monkey at level of VIIIth nerves in a case in which left labyrinthectomy was performed with no resulting degeneration of nerve trunk. Weigert preparation.

rinthectomy, the ipsilateral VIIIth nerve subsequently degenerated.

June 4, 1934: Left labyrinthectomy was performed as in previous case. The animal developed signs of unilateral vestibular dysfunction comparable qualitatively and quantitatively to those seen in Case 1 and as previously reported.

Forty-eight hours after operation the animal was tested for nystagmus. Postrotatory nystagmus time on right rotation 20 times in 20 seconds averaged seven seconds. Postrotatory nystagmus time on left rotation 20 times in 20 sec-

onds averaged 13 seconds. *Caloric Testing:* No response was obtained in left ear with 50 cc. of ice water. Right ear revealed normal response with 3 to 5 cc. of water at 40° F. *Galvanic Testing:* Ear to Ear: With cathode in left ear, good normal response was obtained at 2 to 3 m.a. With cathode in right ear, good normal type response was obtained at 3 to 4 m.a. Ear to back: With cathode in right ear and anode on back, good normal type response was obtained at 4 to 5 m.a. With cathode on back, good normal type response was obtained 5 to 7 m.a. With cathode in left ear and anode

TABLE III.

NYSTAGMUS REACTION AFTER LEFT LABYRINTHECTOMY
(IPSILATERAL VIIIth NERVE DEGENERATED)

Tests	Procedure	Head Position	Stimulus			Nystagmus
			2 Days After Operation	1 Week After Operation	3 Weeks After Operation	
Galvanic	Right Ear +, Left Ear —	Any	2 to 3 M.A.	3 M.A.	3 M.A.	→
Galvanic	Left Ear +, Right Ear —	Any	3 to 4 M.A.	4 M.A.	3 M.A.	←
Galvanic	Body +, Right Ear —	Any	4 to 5 M.A.	5 M.A.	5 M.A.	←
Galvanic	Right Ear +, Body —	Any	5 to 7 M.A.	5 M.A.	6 M.A.	→
Galvanic	Body +, Left Ear —	Any	6 to 8 M.A.	12 M.A.	20 M.A.	None
Galvanic	Left Ear +, Body —	Any	10 to 12 M.A.	13 M.A.	20 M.A.	None

Note: Reaction to rotation and caloric tests similar to that in Table II.

on back, response was obtained at 6 to 8 m.a. With cathode on back and anode in ear, response was obtained at 10 to 12 m.a.

One Week After Operation: Caloric and rotation tests gave the same results as at the 48-hour stage. Galvanic test revealed at this time the same findings on right as at 48-hour stage, but on left, with the cathode in the ear, a response at 12 m.a., and with cathode on back, a response at 13 m.a.

Three Weeks After Operation: Rotation at this time revealed postrotatory nystagmus time of average eight sec-

onds on right turning 20 times in 20 seconds and average post-rotatory time of 10 to 11 seconds on left turning 20 times in 20 seconds. Caloric test revealed no response from left ear. Galvanic testing revealed, with cathode in ear or with cathode on back on the left, merely a few twitches of eyes but no typical nystagmus at 20 m.a. On the right the responses were as previously found. This relationship of failure of the galvanic response on the left persisted until sacrifice of the ani-

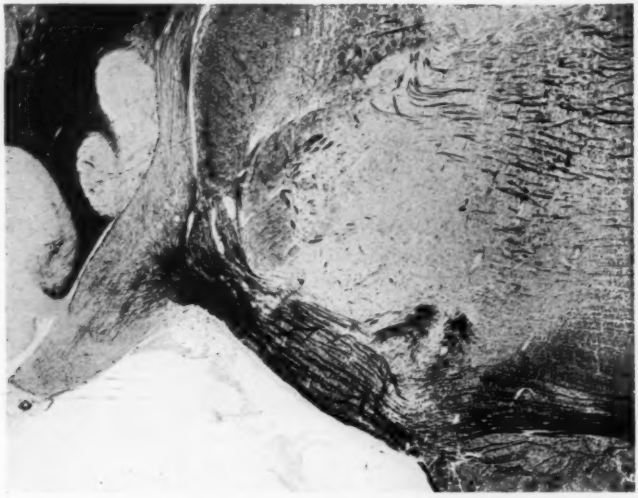

Fig. 2. Weigert preparation of brain stem of a monkey in which left labyrinthectomy was performed several months before sacrifice of animal. The labyrinthectomy resulted in ipsilateral VIIIth nerve trunk degeneration. In this case the galvanic response was negative.

mal at the three-month stage, when the ears and brain were prepared for histological study. Serial section of the temporal bones revealed a complete destruction of the labyrinth on the left and a normal labyrinth on the right. No evidence of Scarpa's ganglion could be found on the left. In addition, study of the Weigert preparations of the brain stem revealed a degeneration of the VIIIth nerve on the left as shown by Fig. 2, which is a microphotograph of a Weigert preparation of the brain stem in this case. As can be seen, the trunk

of the left VIIIth nerve is degenerated. In this case the degeneration of the nerve could be followed to the primary auditory and vestibular ganglia in the brain stem. *General Consideration:* It is of interest in this case, as contrasted to Case 1, that there was a progressing loss of response to the galvanic current on the left despite the fact that in this case, just as in Case 1, a labyrinthectomy had been performed. In this case, however, the labyrinthectomy had apparently gone deeply enough to involve Scarpa's ganglion, with the result that there was a progessing degeneration of the VIIIth nerve on the side of the lesion. Associated with the degeneration of the nerve there occurred a diminishing and finally absent galvanic response on the side of the lesion. This is contrasted to Case 1, in which, associated with a normal VIIIth nerve and destroyed labyrinth, the galvanic response was obtained on the involved side. In both cases the caloric and rotation tests gave evidence of vestibular loss on the left. Thus, Case 2 would seem to indicate that the failure to obtain the nystagmus reaction to galvanic stimulation was caused by degeneration of the VIIIth nerve.

The importance of the VIIIth nerve itself in the galvanic response was tested by cases in which the nerve was cut as follows:

Case 3: Table IV is a table summarizing the results of testing in a monkey with typical intracranial VIIIth nerve section.

May 2, 1934: Section of right VIIIth nerve intracranially. This monkey showed all of the characteristic defects of motion and posture caused by a total unilateral vestibular loss as previously described.

Forty-eight Hours After Operation: Vestibular Tests: Left rotation, postrotatory nystagmus time averaged seven seconds. Right rotation, postrotatory nystagmus time averaged 13 seconds. *Caloric Testing:* Left ear, 5 cc. water at 40° F. produced good, normal, active response. Right ear, no response was obtained with 50 cc. *Galvanic Testing:* Ear to ear: With cathode in left ear good, normal response obtained at 2 to 3 m.a. Cathode in right ear, active, normal response obtained 4 to 5 m.a. Cathode in left ear, anode on back,

TABLE IV.

NYSTAGMUS REACTION AFTER SECTION OF RIGHT VIIIth NERVE.

Tests	Procedure	Head Position	Stimulus	Nystagmus	Stimulus	Nystagmus
			2 Days and 5 Days After Operation		3 Weeks After Operation	
Rotation	Rotation Right	Face Front	20 Times in →	For 13 Secs.	20 Times in →	For 9 Secs.
Rotation	Rotation Left	Face Front	20 Seconds ←	For 7 Secs.	20 Seconds ←	For 8 Secs.
Caloric	Right Ear (40° F.)	Face Down	50 CC.	None	50 CC.	None
Caloric	Right Ear (40° F.)	Face Front	50 CC.	None	50 CC.	None
Caloric	Left Ear (40° F.)	Face Down	5 CC.	→	3 to 5 CC.	→
Caloric	Left Ear (40° F.)	Face Front	5 CC.	←	3 to 5 CC.	←
Galvanic	Right Ear +, Left Ear —	Any	2 to 3 M.A.	→	2 to 3 M.A.	→
Galvanic	Left Ear +, Right Ear —	Any	4 to 5 M.A.	←	3 to 4 M.A.	←
Galvanic	Body +, Left Ear —	Any	4 to 5 M.A.	→	4 to 5 M.A.	→
Galvanic	Left Ear +, Body —	Any	4 to 5 M.A.	←	4 to 5 M.A.	←
Galvanic	Body +, Right Ear —	Any	4 to 20 M.A.	None	4 to 20 M.A.	None
Galvanic	Right Ear +, Body —	Any	4 to 20 M.A.	None	4 to 20 M.A.	None

good, normal response obtained 3 to 4 m.a. Cathode on back, normal response obtained 5 to 7 m.a. Right ear, cathode in ear, anode on back, few twitches obtained at 12 to 15 m.a. Cathode on back, anode in ear, few twitches obtained 15 to 20 m.a. One week after operation the findings were the same.

Three Weeks After Operation: Postrotatory nystagmus time on left rotation averaged nine seconds; on right rotation, nine seconds. No response obtained from right ear with large

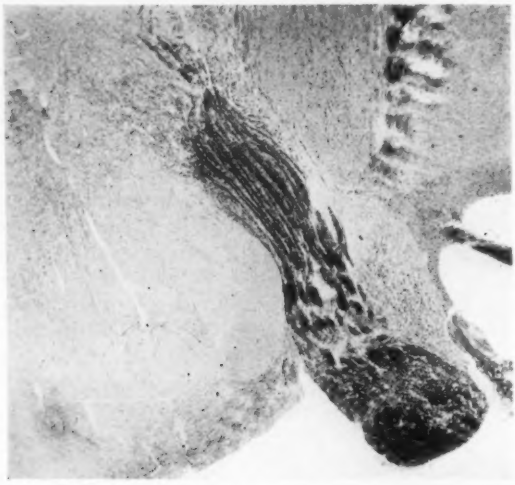

Fig. 3. Marchi preparation of frontal section of a monkey in which the right VIIIth nerve was sectioned three weeks before sacrifice of the animal. The degenerating nerve stump can be followed into the primary vestibular nuclei. Galvanic response was absent in this case.

amount of ice water. Good, normal response obtained from left ear with 3 to 5 cc. With the galvanic current the same results as previously obtained from the left ear, whereas on the right only a few twitches were obtained at 20 m.a. with cathode in ear or on back.

The animal was sacrificed on this date for study with the Marchi method and sections of the brain stem revealed a degeneration of the VIIIth nerve trunk in both of its divisions. The degenerating fibres could be followed easily to the

primary auditory and vestibular centres in the medulla. Sections of the ear revealed a progressing degeneration of the structures of the ear, particularly the cochlea, in which a gradual disappearing of the cells of the spiral ganglia could be seen to be occurring. Fig. 3 shows a Marchi preparation of the brain stem in this case, demonstrating the degenerating fibres in the vestibular division as they bend into the brain stem to enter the primary vestibular centres.

General Impression: All cases in which the VIIIth nerve was sectioned unilaterally intracranially revealed an absent galvanic response on the side of the section associated with an absent caloric response and the abnormal rotation of the early stage. In some cases, especially where little attempt was made to separate the two ends of the cut nerve stem, a few twitches were obtained from the side of the section with a fairly high current in the very early stage following the operation. The response disappeared completely, however, by the chronic stage and was always absent in those cases in which an attempt was made to separate the ends of the nerve. It would seem from this type of case that the nerve action has something to do with the production of a galvanic nystagmus. However, the question as to the participation of the nerve was made easier to answer in light of an experiment in which bilateral labyrinthectomy was produced with resulting VIIIth nerve degeneration on one side and preservation of the VIIIth nerve on the other side as follows:

Case 4: Table V is a chart summarizing the results of testing in this case.

June 1, 1933: Bilateral labyrinthectomy. Following the operation the monkey revealed definitely the clinical evidence of total loss of vestibular function on both sides as described by us in a previous paper.

Forty-eight hours after operation the vestibular tests revealed the following: *Rotation:* No postrotatory nystagmus was elicited following the usual rotation in either direction. *Caloric:* No nystagmus was elicited from either side even with large quantities of ice water. *Galvanic:* Ear to ear: With cathode in either ear and anode in the opposite ear good, normal typical nystagmus response was obtained at 3 to 4 m.a. *Ear to Back Testing:* Right ear: With cathode

in the ear, normal type nystagmus response was obtained at 4 to 5 m.a. With cathode on back, normal type nystagmus obtained at 6 to 7 m.a. Left ear: With cathode in ear normal type nystagmus response was obtained at 5 to 6 m.a. With cathode on back, normal type nystagmus response was obtained with 6 to 7 m.a.

One Month After Operation: Vestibular tests revealed the following: *Rotation Test:* No postrotatory nystagmus following rotation in either direction. *Caloric:* No nystagmus

TABLE V.

NYSTAGMUS REACTION AFTER BILATERAL LABYRINTHECTOMY (VIIIth NERVES: RIGHT NORMAL. LEFT DEGENERATED).

Tests	Procedure	Head Position	Stimulus			Nystagmus
			2 Days After Operation	1 Month After Operation	2 Months After Operation	
Galvanic	Right Ear +, Left Ear —	Any	3 to 4 M.A.	8 M.A.	9 M.A.	→
Galvanic	Left Ear +, Right Ear —	Any	3 to 4 M.A.	4 M.A.	4 M.A.	←
Galvanic	Body +, Right Ear —	Any	4 to 5 M.A.	4 M.A.	4 M.A.	←
Galvanic	Right Ear +, Body —	Any	6 to 7 M.A.	6 M.A.	7 M.A.	→
Galvanic	Body +, Left Ear —	Any	5 to 6 M.A.	14 M.A.	20 M.A.	None
Galvanic	Left Ear +, Body —	Any	6 to 7 M.A.	16 M.A.	20 M.A.	None

Note: No reaction on rotation or caloric tests.

obtained from either ear even with large quantities of ice water. *Galvanic:* Ear to ear: With cathode in right ear, normal type response obtained at 4 m.a. With cathode in left ear, normal type response obtained at 8 m.a. Ear to back: Right ear, with cathode in ear, normal type response obtained at 4 m.a. With cathode on back, normal type response obtained at 6 m.a. Left ear, cathode in ear, few twitches obtained at 14 m.a. With cathode on back, few twitches obtained at 16 m.a.

Two Months After Operation: Vestibular reactions were as follows: Rotation and caloric methods elicited no nystag-

mus from either ear. *Galvanic Test:* Ear to ear: Cathode in right ear, there was a normal type response at 9 m.a. *Ear to Back Testing:* Right ear, cathode in ear, there was a normal type response at 7 m.a. Left ear, cathode in ear, there were a few twitches but no definite nystagmus at 15 to 20 m.a. Cathode on back, there were a few twitches but no nystagmus at 20 m.a. The animal was sacrificed and the temporal bones and brain stem were prepared for serial study. Study of the sections of the temporal bone revealed on the

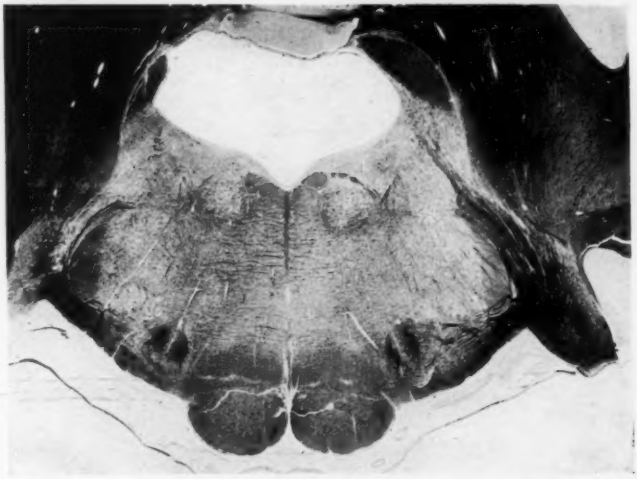

Fig. 4. Weigert preparation of frontal section of a monkey, in which bilateral labyrinthectomy was performed, resulting in degeneration of the left nerve trunk and preservation of the right. No responses to rotation or caloric testing obtained. Galvanic response negative on left, positive on right.

right that the labyrinthectomy had been superficial in type. Much of the cochlea remained undamaged and the vestibular end-organ remained intact. The vestibular ganglion could be seen and was found to be relatively intact. On the left, on the other hand, the labyrinthectomy was complete. Sections revealed merely a large mass of scar tissue. No evidence of cochlear, vestibular or vestibular ganglion structures could be made out. There appeared to be possibly a few nerve fibres and questionably a few cells in the remnants of the vestibular ganglion on this side. Section of the brain stem (see Fig. 4)

revealed a practically complete degeneration of the trunk of the VIIIth nerve on the left. On the right the nerve appeared to be well preserved.

In this case it would appear again as if the presence of a positive galvanic response was associated with the intactness of the VIIIth nerve, because on the left, where the VIIIth nerve was degenerated except for merely a few fibres, a galvanic response could be elicited only with the highest current of 20 m.a., and then only in the form of a few jerks, which did not constitute a real nystagmus. On the right side, on the other hand, a good normal response could be elicited throughout the whole course of study and here the lesion was found to involve merely a portion of the labyrinth with good preservation of the vestibular ganglion and nerve trunk.

Case 5: Table VI is a chart summarizing the results of testing in this case.

Sept. 2, 1934: First operation, puncture of the round-window of the ear. Following the usual procedure used in a radical mastoidectomy the promontory was exposed on the right side and the round-window membrane was punctured with a fine needle. Inasmuch as this monkey was being used for a different purpose at the time, studies in the acute phase were not made with the galvanic test. Rotation and caloric tests, however, gave results indicating acute unilateral peripheral dysfunction. This monkey showed very definitely a progressing vestibular loss on the side of the operation. By one month after the operation the head posture and other features of unilateral loss of vestibular function were present. At this time nystagmus tests gave the following results: *Rotation Test:* Right turning 20 times in 20 seconds, average time postrotatory nystagmus 10 seconds; left turning 20 times in 20 seconds, postrotatory nystagmus time nine seconds. No response was obtained with ice water in the right ear, even in large quantities. Five cc. of water in the left ear gave a good, normal, typical response at 3 to 5 cc. *Galvanic Tests:* Ear to ear, good normal response was obtained with the cathode in either ear and the anode in the opposite ear at from 3 to 5 m.a. Ear to back and back to ear stimulation on either side gave essentially the same and equal values, all of which were normal. The findings suggested, although the right end-organ was supposedly ablated, that the VIIIth nerve

TABLE VI.

NYSTAGMUS REACTION AFTER PUNCTURE OF RIGHT ROUND-WINDOW AND LATER SECTION OF LEFT VIIIth NERVE (LEFT VIIIth NERVE DEGENERATED, RIGHT NORMAL).

Puncture of round-window with a fine needle, avoiding as much as possible trauma to the inner ear. Spontaneous symptoms similar in type but less in degree to those following unilateral labyrinthectomy or section of VIIIth nerve, i.e., nystagmus to side of normal ear and forced movement to side of ear lesion. One month after puncture of right round-window, section of left VIIIth nerve was done.

Tests	Procedure	Head Position	Stimulus	Nystagmus
ONE MONTH AFTER RIGHT ROUND-WINDOW PUNCTURE				
Rotation	Rotation Right	Face Front	20 Times in	→ For 10 Seconds
Rotation	Rotation Left	Face Front	20 Seconds	← For 9 Seconds
Caloric	Right Ear (40° F.)	Face Down	50 CC.	None
Caloric	Right Ear (40° F.)	Face Front	50 CC.	None
Caloric	Left Ear (40° F.)	Face Down	3 to 5 CC.	→
Caloric	Left Ear (40° F.)	Face Front	3 to 5 CC.	←
Galvanic	Right Ear +, Left Ear —	Any	3 to 5 M.A.	→
Galvanic	Left Ear +, Right Ear —	Any	3 to 5 M.A.	←
Galvanic	Body +, Right Ear —	Any	4 to 5 M.A.	←
Galvanic	Right Ear +, Body —	Any	4 to 5 M.A.	→
Galvanic	Body +, Left Ear —	Any	4 to 5 M.A.	→
Galvanic	Left Ear +, Body —	Any	4 to 5 M.A.	←
AFTER LEFT VIIIth NERVE SECTION				
Galvanic	Right Ear +, Left Ear —	Any	7 to 8 M.A.	→
Galvanic	Left Ear +, Right Ear —	Any	3 to 5 M.A.	←
Galvanic	Body +, Right Ear —	Any	4 to 5 M.A.	←
Galvanic	Right Ear +, Body —	Any	7 to 8 M.A.	→
Galvanic	Body +, Left Ear —	Any	20 M.A.	None
Galvanic	Left Ear +, Body —	Any	20 M.A.	None

Note: No nystagmus reaction on rotation or caloric tests.

was still intact and in order to compare the two sides the second operation was performed.

Oct. 7, 1934: Section of the left VIIIth nerve intracranially. Forty-eight hours after operation induced nystagmus observations as follows: *Rotation Test:* No postrotatory nystagmus after right or left rotation. *Caloric Test:* No response was elicited in either ear with large quantities of water at 40° F. *Galvanic Test:* Ear to ear testing with cathode in left ear gave a normal, typical response at 7 to 8 m.a. With cathode in right ear the response was produced at 3 to 5 m.a. *Ear to Back Testing:* Right side, with cathode in ear, good, typical nystagmus response was obtained at 4 to 5 m.a. With cathode on back, normal type response obtained at 7 to 8 m.a. Left ear, no response was obtained up to 20 m.a. either with cathode in ear or cathode on back. Two weeks and one month after the second operation, nystagmus tests gave the same results as after 48 hours. Study of the left ear, the side of the nerve section, revealed a complete disappearance of the cells of the spiral ganglia. On the right side there was a general shrunken appearance of the cochlea with, however, preservation of the spiral ganglia and of the vestibular end-organs, which appeared, however, to be somewhat shrunken or atrophic. On this side the vestibular ganglion could be seen and it appeared to be generally normal. Section of the brain stem revealed a complete degeneration of the trunk of the VIIIth nerve on the left and the degeneration could be followed as far as the primary cochlear and vestibular centres. On the right side both divisions of the VIIIth nerve appeared intact.

This case reveals the importance of the galvanic test, in that it demonstrates a negative result with a nonfunctioning nerve and its positivity with a functioning nerve, despite a clinically nonfunctioning end-organ.

DISCUSSION.

The findings reported here of the nystagmus reaction in monkeys with peripheral vestibular lesions to the rotation, caloric and galvanic tests consistently have been present in several similar cases studied.

For a brief period only, after unilateral labyrinthectomy or intracranial section of the VIIIth nerve, was the post-

rotatory nystagmus time shorter following turning in the direction of the normal ear than it was in the direction of the operated side. From a week to a month after those vestibular lesions the postrotatory nystagmus became equal in time following right and left rotation and the duration was shorter than that in the normal monkey in most of the cases studied. Although similar observations repeatedly have been reported on lower forms of mammals than the monkey, and although it was stated by Ruttin[7] some 20 years ago that the postrotatory nystagmus time was the same following right and left rotation in patients with the loss of function of one vestibular end-organ from infection, it is common practice still to use the rotation test for diagnostic information in unilateral vestibular disease. The following case of VIIIth nerve tumor is cited as one of the many patients examined who had either labyrinthitis, trauma of the inner ear or disease of the VIIIth nerve intracranially, in whom the rotation test did not disclose the unilateral vestibular loss of function that was present.

A woman, age 30 years, was admitted to the Neurological Institute, complaining of tinnitus and loss of hearing in the right ear for three years. There was no history of vertigo or middle ear infection. She had received inflations of the Eustachian tube, treatment for sinusitis and electrotherapy. There had been no relief of her symptoms. A neurological examination just prior to the functional ear examination recorded here (see Fig. 5) revealed evidence suggesting a right VIIIth nerve tumor. Hearing was greatly impaired in the right ear. Vestibular reactions were absent on the right side and impaired on the left. The cochlear and vestibular impairment here confirmed the diagnosis of an VIIIth nerve tumor, which was found on operation.

The examination of this patient illustrates the lack of dependability of the rotation tests in revealing unilateral vestibular loss in VIIIth nerve tumors. A record of the audiometric test and rotation test, which were done two and a half years ago, was available and showed findings almost identical with those shown here. No caloric test was done at that time. It is a fair assumption that a caloric test on the initial examination would have given evidence of sufficient vestibular impairment on the right side, which, along with the cochlear

loss, would have prompted a neurological investigation of this patient's condition. We should bear in mind that the early subjective disturbances of VIIIth nerve tumors are almost invariably deafness, tinnitus and vertiginous attacks. The vertigo may be very slight or entirely absent, as in this case. The histories of 30 patients we examined with unilateral tumors involving the VIIIth nerve did not show even one in which the

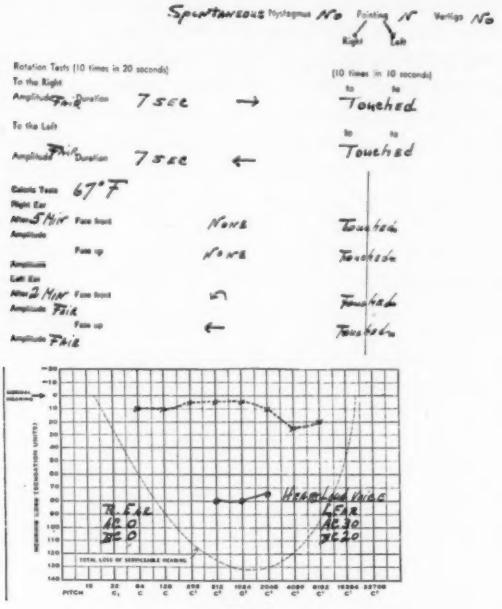

Fig. 5.

disease was suspected by the otologists; although all of them had received treatment from one to three years for unilateral deafness and tinnitus and some for vertigo. Our experience warrants the opinion that all patients with unilateral loss of hearing, not having a manifest suppurative middle ear infection to account for it, should have the caloric test along with the hearing tests before a diagnosis is made.

All of us are familiar with the use and claims for the reactions to the rotation test in the selection of aviation personnel.

The reactions, however, in the course of time have been found to be of such little value that the Army and Navy give little importance to them. The Bureau of Air Commerce has never made the rotation test a part of the examination for civilian fliers, but use such tests as the Romberg, standing on one foot with the eyes closed and walking a straight line or circle with the eyes closed to disclose manifest disturbances of equilibrium. A man who had been a ballroom dancer for several years was examined as a prospective aviator. He could stand with eyes closed on one foot without any unsteadiness for minutes. He volunteered to have the rotation, caloric and galvanic tests. On the rotation test of turning 10 times in 20 seconds there was no nystagmus, vertigo or pastpointing. On swift turning there were a few postrotatory nystagmoid jerks of the eyes. As he showed also such considerable lack of reactivity on the caloric and galvanic tests, it was assumed that his tolerance was a central nervous system phenomenon. Although this applicant would not be aware of angular movements so acutely as the normal, also he would not suffer of vertigo from maneuvers that would cause such in the normal. Since an aviator relies so greatly on his vision to maintain his equilibrium, the degree of acquired tolerance in this man for angular movements is likely to be more beneficial than harmful to him.

Although regularly we have found a difference in the right and left postrotatory nystagmus times in the acute stage after unilateral vestibular lesions, and in a majority of the cases a reduction but equalization in the postrotatory nystagmus times in the chronic stage, it is not so dependable as the caloric test, which is suitable for all patients having unilateral peripheral vestibular loss from end-organ lesions. In addition to this opinion, we believe that the rotation test should be included in the routine functional ear examination because frequently reactions are brought out that are of importance in the diagnosis when considered with other neurological symptoms present. In the reviewing of some 2,000 records of functional ear examinations, it was noted that a prolonged postrotatory nystagmus time more frequently was seen in patients with the diagnosis of some degenerative brain stem lesion than in those with any other disease. The postrotatory nystagmus time was prolonged

to over 30 seconds in a majority of the patients with the diagnosis of multiple sclerosis.

In Table VI there is stated the spontaneous vestibular phenomena following a small puncture of the round-window membrane. The nystagmus and forced movements were similar in type, but milder than those following labyrinthectomy or section of the VIIIth nerve. All of us know that when patients with middle ear and mastoid infections manifest a disturbance of vestibular function, the life of the patient may depend upon treatment being directed by a correct diagnosis. It is important to recognize whether the vestibular disturbances are due to a so-called irritative condition or an infection of the inner ear, or whether they are due to some intracranial complication of the ear condition, such as a brain abscess or localized meningitis. Valuable information may be had by correctly interpreting the significance of the spontaneous nystagmus, vertigo and forced movements, as well as evaluating the results of the cochlear and vestibular tests. Several years ago, when on the house staff at Bellevue Hospital, it was usual after a radical mastoidectomy to observe a nystagmus to the side of the good ear for 24 to 48 hours. Of course, the vertigo was in the direction of the nystagmus and forced movements opposite. Although this vestibular disturbance appeared to be due to the trauma of the operation and a good example of an irritative cause, the direction of the nystagmus and forced movements did not conform to the textbook description of an irritative disturbance of the vestibular function. The following description of the spontaneous phenomena in infections involving the inner ear is that commonly seen in all textbooks and in other publications, with one exception, which have come to our attention. "The characteristic functions of serous labyrinthitis are the prolongation of the irritative stage and the incompleteness of the paretic stage, and the usual nystagmus towards the affected side, a nystagmus of irritation. Diffuse suppurative labyrinthitis is characterized by destruction. *1.* A stage of irritation where all signs are produced by the affected labyrinth, intense vertigo, nystagmus, often in both directions, but stronger to the diseased side, with the slow movements of the eyes, falling and pastpointing to the healthy side, with rapid and complete loss of hearing. This state may last for a few days; in sudden invasions it may pass

so quickly that it is undetected. *2.* A stage of suppression when all signs are due to the uncompensated activity of the surviving labyrinth. The nystagmus swings over and is directed to the healthy side, falling and pastpointing to the diseased side. Hearing on that side is abolished, and on that side the labyrinth tests are negative, except the galvanic. *3.* A stage of compensation—there is complete loss of hearing on the diseased side, and no response to caloric or fistula tests, but the rotation tests may give a normal response (Ruttin's reaction)."[8]

The one exception, we have read, to this characterization of the vestibular disturbance in infections involving the inner ear is the following by Wilson:[9] "In lesions of the peripheral organ of the ear, the spontaneous vestibular reflexes observed are to be regarded as due to a loss of function and not to an increase of function, supposedly owing to irritation. If such an increase of function occurs, it must be rare, as I have never observed it. It would appear as a deviation of the eyes to the side opposite the lesion. Following a mastoid operation, especially in elderly people in whom the bone is sclerotic, one occasionally sees a nystagmus lasting 24 to 48 hours, which passes off without further disturbance. This, I believe, is due to labyrinthine disturbance and might be called an irritative lesion. But in no case have I seen the eyes deviated to the opposite side; the slow component is always to the side of the lesion, a depressive effect. After experimental destruction of the labyrinth in whole or in part in animals, as soon as the animals recover from the anesthetic the spontaneous nystagmus observed indicates loss of function. Clinical cases have been recorded in which failure to recognize this has been followed by unfortunate results. In one such case following acute inflammation of the left ear there was a slow deviation to the right and a quick return to the left; this was diagnosed as due to inflammatory involvement of the left labyrinth and not till five weeks later, when choked disc resulted, was the patient operated on for left cerebellar abscess."

Our observations are in agreement with those of Wilson. The following three cases are cited, as all showed early after the onset of the vestibular disturbances the phenomena usually characterized as representing a manifest diffuse sup-

purative labyrinthitis, or some other destructive inner ear lesion, and the patients recovered, having normal cochlear and vestibular reactions. If it be contended that the nystagmus may have been to the side of the diseased ear before our observation, still the recovery of cochlear and vestibular functions warrants the conclusion that the labyrinth lesion was not a destructive one.

Case 1: A woman, age 30 years, was admitted to the Presbyterian Hospital suffering from the usual symptoms of right acute mastoiditis. There was no manifest disturbance of vestibular function. It was a recurrent ear infection. On removing a polyp from the middle ear the patient fell to the floor from the vertigo she developed. When examined four hours later she had intense vertigo with vomiting and was lying on the left side with her eyes closed. There was a horizontal-rotatory nystagmus to the left, large amplitude and the pastpointing was in the opposite direction. The right ear was irrigated with water at 110°, which reversed the direction of the spontaneous nystagmus. She could hear loud spoken voice on the right side. After a week's rest in bed the vestibular symptoms subsided and a simple mastoidectomy was performed. She made an uneventful recovery and the cochlear and vestibular reactions were normal.

Case 2: A woman, age 24 years, was admitted to the Presbyterian Hospital with an acute suppurative right middle ear infection of two days' duration. She complained of intense pain in the right side of her head. She had nausea and vomiting, nystagmus to the left and falling to the right. Her temperature was from 100° to 102.5°. Roentgen examination showed cloudiness of mastoid cells. She was not totally deaf on the affected side and warm water changed the direction of the nystagmus. In spite of her distressing vestibular disturbance, it was not considered advisable to perform any operation on the mastoid so early after the onset of the infection. The symptoms persisted and a simple mastoidectomy was performed eight days after the middle ear infection began. Recovery was uneventful. The cochlear and vestibular reactions were normal.

Case 3: A woman, age 18 years, was admitted to the Presbyterian Hospital with mild symptoms of brain concussion and bleeding from the right middle ear from an injury in

an automobile accident. She had vertigo and the nystagmus was towards the healthy side. Her hearing in the injured ear was about three feet for whispered voice. She recovered and had normal cochlear and vestibular reactions.

If we had believed that the spontaneous vestibular phenomena in these patients indicated an invasion of the inner ear by an infection in the first two patients as the usual description warrants, an investigation of the spinal fluid would have been in order. This was not done in either one. In the presence of an infection of the inner ear there is no definite evidence that the reduction of the spinal fluid pressure does not increase the chances of an extension of the infection to the meninges, so a lumbar puncture should not be done without a reasonable indication. We never have seen a patient with an ear infection causing a disturbance of vestibular function develop an intracranial complication through the inner ear, without first there being a total loss of reactivity to cochlear and vestibular tests. The functional ear examination would seem to be our best gauge in estimating the disease of the inner ear and determining when a spinal fluid examination is indicated, rather than the direction of the nystagmus, vertigo and pastpointing. A well sustained nystagmus, with the eyes in the primary position, towards the side of a proven uncomplicated ear infection never has been seen by us. It is our opinion that when the nystagmus does occur in the direction of the infected ear a satisfactory investigation for an intracranial complication is indicated before any operative procedure on the ear is warranted.

CONCLUSIONS.

1. The caloric test should be used routinely in the examination to determine unilateral vestibular loss of function, because the reactions to the rotation test are not dependable. The reactions, however, to the rotation test *for a variable short time* after a total unilateral vestibular loss may suggest impairment of function on the side of the lesion.

2. The galvanic test, with the electrodes placed in the hand and in the ear, should be used when there is no response to the caloric test. Normal reactions to the galvanic current indicate that the VIIIth nerve has not degenerated.

3. The direction (quick component) of the nystagmus commonly is towards the side of the normal ear in the presence of vestibular disturbances, due to an ear infection or trauma, and may be due to an irritative lesion as well as to a destructive lesion of the inner ear. The knowing of the cochlear and vestibular function present from the reactions to testing in patients with manifest disturbances of vestibular function from infection or trauma is of greater diagnostic value than is the direction of the spontaneous nystagmus, vertigo and pastpointing.

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20 East 53rd Street.

REPORT OF BOARD OF TRUSTEES OF RESEARCH FUND.*

DR. JOHN B. RAE, New York, and
DR. J. GORDON WILSON, Chicago.

The Annual Meeting of the Trustees of the Research Fund was held at the Academy of Medicine, 2 East 103rd street, New York, on Saturday, Jan. 9, 1937.

The obituary notice on the death of Dr. Arthur B. Duel was presented by Dr. John R. Page, and was ordered spread in full upon the minutes.

The Board then elected the following officers: Chairman, Dr. J. Gordon Wilson, Chicago; Vice-Chairman, Mr. Starling W. Childs, of New York; Treasurer, Dr. Edmund P. Fowler, of New York; Secretary, Miss Mary R. Taylor.

As lay advisers, the Board is fortunate to retain the services of Mr. George E. Coleman, of Santa Barbara, Calif., and Mr. W. J. Dupratt White, of New York City.

As professional advisers, the Board is also fortunate to retain the services of Dr. Madison Bentley, Sage Professor of Psychology at Cornell University; of Dr. W. A. Neilson, President of Smith College, and of Dr. John Tait, Professor of Physiology, McGill University.

Dr. Wesley C. Bowers, of New York City, was elected a Trustee to fill the vacancy resulting from the death of Dr. Arthur Duel.

Dr. J. Gordon Wilson presented his epitome of the reports of the research workers, all of which had been submitted to individual members of the Board prior to the meeting.

On motion the Board thanked Dr. Wilson for his able resumé and empowered him to thank the research workers for their valuable reports.

The thanks of the Board for interest and services were extended to Mr. Childs, who responded, and the Chairman

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*Read at the Seventieth Annual Meeting of the American Otological Society, Long Beach, N. Y., May 28, 1937.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 17, 1937.

was authorized to write, expressing similar thanks to Mr. J. Dupratt White, who was unavoidably absent.

During the year the third volume on the literature on otosclerosis was published, bringing the work up to 1935. Dr. F. Robbins wrote the text and the annotated bibliography. This work continues.

Estimated income for 1937 amounted approximately to $14,700, and allocations were made to the amount of $14,650.

Since the Annual Meeting in January—and therefore not a part of its proceedings—the Trustees report with intense gratification a gift to the permanent fund of approximately $100,000. The donor is the Vice-Chairman of the Board, Mr. Starling W. Childs, and this latest gift is concrete evidence of the interest and service which he has previously proved in the work of this investigation. In making this contribution to the capital fund, Mr. Childs makes certain reservations as to the immediate disposition of income. These conditions will be scrupulously observed by the Board, but it must be a source of gratification to all concerned that in later years and, as our knowledge increases, he will still be contributing toward the solution of the difficult problem of chronic progressive deafness.

REPORT OF THE NEW YORK COMMITTEE ON OTITIC MENINGITIS.*

DR. J. G. DWYER, New York.

The New York Committee on Meningitis has been carrying on the work during the last year and in making its annual report would point out that, for the first time, we have been able to report progress in several directions, which will be elaborated upon further.

During the last year the literature everywhere has been full of reports covering cases of otitic meningitits, with a larger percentage of recoveries than ever before. These reports have been very complete and the bacteriological work has been exact. This is a distinct step forward, as the bacteriological diagnosis in the past has been very confusing and unsatisfactory. The use of sulfanilamide in its various forms and the combined use of this drug with allied substances has been a new development, which will be gone into later in this report, in some detail.

The Committee received reports with complete records covering 42 cases. In addition, we had knowledge of over 30 other cases reported indirectly to us, but upon which we did not pass officially. The Board of Health of the City of New York also had a large series that they reported on at the Academy of Medicine. Dr. D. Cunning had nine cases not reported upon in this series, but reference to the same is made below. Thus, one can see at a glance how important the question of otitic meningitis is year by year. It is easily the most important problem confronting us, dealing, as it does, with life and death.

In addition to the points in diagnosis stressed in other years, we wish, from our experience, to emphasize three very important points that have helped us in the exact diagnosis to clear up some obscure points. These are the blood counts,

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the bacteriology and the significance of a persistently cloudy spinal fluid without organisms. These points have been dealt with in former reports and are here mentioned only for the sake of emphasis. The recognition of the fact that a persistently cloudy fluid, without organisms, means that the infection is still extradural and is probably due to a sequestrum, or a localized collection of pus, has saved several lives in this series.

SULFANILAMIDE AND ITS RELATED COMPOUNDS.

The use of the various forms of sulfanilamide and its related compounds has given the greatest hope we have had in years in the treatment of streptococcus hemolyticus meningitis. The reports from all over the country are very encouraging, but a note of warning should be injected at this stage of a new therapy, as a swing of the pendulum may go too far one way, which has been the history of most new drugs. They have been hailed as "cure-alls" and then discarded entirely. Hence, it is important that the contraindications and dangers may be recognized. Most patients show no unfavorable effects from the use of this drug and its related compounds, but there are reports of complications that may arise, or be attributed to it. A patient with a nephritis may have the nephritis increased by its use; other patients may develop a transitory albuminuria. There are some reports of acute yellow atrophy, and in some cases an anemia with the formation of methemoglobin; hence, the promiscuous use of the drug must be deprecated. Acidosis is reported in other cases. The complications that may arise must be kept in mind.

METHOD OF ACTION OF SULFANILAMIDE.

How this drug and its allied compounds work has been a matter of conjecture. Most authorities have believed that they have a germicidal or disinfectant effect. Dr. F. Gay, Professor of Bacteriology at the College of Physicians and Surgeons, Columbia University, has found that the action of the drug is through stimulation of the mononuclear leukocytes. This report is ready for publication and the research work has been done under a grant from The Philip Hanson Hiss, Jr., Memorial Fund. It is a coincidence that the late Dr.

Hiss had devoted his life to research on the relation of the blood cells to infections, and he believed that the main defensive factors included the large mononuclear leukocytes. The writer wishes to thank Dr. Gay for permission to report the above findings of the action of sulfanilamide.

METHOD OF USE.

There is a question as to the form of the drug to be used and the dosage. There are several forms in which it can be used and in some cases one can combine the various forms. Thus, one can use the several preparations of sulfanilamide alone by mouth or combine the simple form with the dye-radical "prontosil," which may be given intramuscularly. Also, you can use the ordinary sulfanilamide by mouth, combined with the crystalline form by the subcutaneous intramuscular or spinal route. "Prontosil" is not given intravenously or intraspinally. The crystalline form is dissolved in 0.8 per cent saline. In some desperate cases the sulfanilamide has been given orally; the "prontosil" intramuscularly and the crystalline solution intraspinally.

Naturally, with a new drug like the above, there is a question as to the proper form to use, the dosage, etc. Some recommend especially large dosage; others the smaller dosage. Undoubtedly these questions will be solved as we get more experience with these forms of medication. Dr. Josephine Neal, of the New York Board of Health, reported a series of 11 cases of streptococcus hemolyticus meningitis treated with this drug, with nine recoveries. Dr. Daniel Cunning, of the Queensboro Hospital and the Manhattan Eye, Ear and Throat Hospital, reported a series of nine cases, with six recoveries. These cases are not included in our series. These results are startling when you consider the reports of previous years.

Dr. Neal recommends 5-20 cc. of the "prontosil" every four hours, the dose depending on the size of the patient, until the temperature has declined. At the same time one to three of the 5 gr. tablets should be given orally every six hours. The use of the oral administration should be continued for at least a week to 10 days after the temperature has become normal. Dr. Cunning in his series in some cases opened

the dura, put the patient in the Trendelenberg position and used the drug freely. In some cases he gave the crystalline form in the spinal fluid.

The future looks a little brighter for the solution of this problem, but as we have followed the rainbow so long we naturally have to go slowly before we come to any conclusion. This report is made for the New York Committee by its Chairman and the latter wishes to acknowledge the hearty co-operation of all its members.

REPORT OF THE CHICAGO COMMITTEE ON OTITIC MENINGITIS.*

DR. ALFRED LEWY and DR. ELMER W. HAGENS, Chicago.

PART I—DR. ALFRED LEWY.

In considering the diagnosis of meningitis for the purpose of treatment, we are confronted with the fact, first, that it may be of nonsurgical type, meningococcic, tubercular, influenzal, aseptic lymphogenous, or due to encephalitis or neoplasm. Secondly, if associated with a discharging ear, in which event, unless promptly proved otherwise, we assume for purposes of treatment that the suppurating ear is causative, we still do not know whether the meningitis is generalized (by which I mean that viable organisms may be cultured from the spinal fluid), or still localized and secondary to sinus thrombosis, extradural abscess or brain abscess, or merely so-called meningismus (a term that I deplore), thus offering hope for successful surgery. We, therefore, recommend the following procedure:

1. When the patient is seen with a stiff neck, Kernig sign and fever, perhaps vomiting, without waiting for stupor, delirium, pareses or pathologic reflexes, as complete a history as possible is promptly obtained; evidences of otogenic or rhinogenic origin are carefully looked for; a complete neurological examination is made, followed by spinal puncture (unless definitely contraindicated by brain tumor or abscess).

2. If there is no otitis media and no manifest rhinogenic cause for the meningitis, no surgery is considered as immediately indicated; the patient is studied further for a definite diagnosis, and meningococcic antitoxin (not serum) is given intravenously unless the epidemic type has been definitely ruled out.

3. If there is a discharging ear, barring cases in *extremis*, without waiting for the result of the spinal fluid culture, we proceed immediately to the exenteration of the mastoid,

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with uncovering of the dura in the middle and posterior fossae over an area of about 2 cm. each. This is to explore for possible extradural abscess, etc., and perhaps it also breaks up possible infective communications between the bone and dura. If the epidemic type has not been definitely excluded, the antitoxin is also given intravenously.

How radical this operation should be, whether or not the petrosa should be explored and by what route, likewise the lateral sinus, and whether or not the labyrinth should be exenterated, depends upon the indications in the individual case, and for lack of time consideration of these problems cannot be gone into now.

Whether or not surgery is indicated after a positive culture has been obtained from the spinal fluid under proper technical conditions depends upon the infecting organism. Streptococcus hemolyticus (especially the beta strain) seems to offer more hope than before the use of the sulfonate dye (prontosil). Any pneumococcus is practically hopeless at present. On the other hand, in support of the recommendation not to await the result of culture, I wish to report three representative cases:

H. L., age 28 years, entered hospital Oct. 11, 1936; discharging left ear 24 years, following scarlet fever; headache five weeks, vomiting three days, facial paralysis, left, 10 days, fever two days. Foul discharge, left; right ear very deaf, but negative membrana tympani; stupor, stiff neck, temperature 103°, pulse 100, marked pallor; condition poor. Hemoglobin 85 per cent, reds 4,620,000, whites 16,100. Spinal fluid cloudy, 3,100 cells per c.mm. All polynuclears.

Operative Findings: Perisinus abscess, sinus thrombosis. Jugular ligation. Blood culture negative; spinal fluid culture (reported after operation) Gram positive diplococci; subculture yielding only staphylococcus albus. On investigation it appeared that spinal tap was done without gloves and without flaming cotton stopper of culture tube. The culture report is therefore set aside as unreliable. Patient discharged Dec. 1, recovered except as to facial paralysis.

E. W., age 11 years, entered Children's Hospital Dec. 28, 1936, with history of discharging ear three weeks; pain back of neck one week. Temperature 99°; stiff neck; Brudzinski; spinal fluid pressure increased, cells 8,850 per c.mm., sugar 83, chlorides 705. Diagnosed by neurological department as otitic

meningitis. Seen by us Dec. 30. Conjugate deviation right; upward nystagmus on looking up; asynergia and adiodokokinesia left arm; slow cerebration; papilloedema left eye. Diagnosis, lesion left posterior fossa, probably abscess. Operation revealed large extradural abscess which was widely uncovered. The sinus could not be identified except beyond the pachymeningitis area, where it was punctured with a thin-bladed knife above and below, free bleeding being obtained. The dura was not incised. Wound packed and left open. Marked but temporary improvement. Jan. 22, 1937, choking of left disk increased and mental hebetude returned. Wound reopened, and after diathermia of dura the Cushing needle obtained pus at depth of 1 cm. anterior to sinus. Needle tract diathermized and small rubber tube inserted, stitched to dura. Recovery. Patient still appears well and all neurological disturbances have disappeared.

C. S., age 18 years, admitted to Contagious Hospital, Aug. 9, 1936, as a suspect epidemic meningitis with classical symptoms, where it was discovered that he had an otitis media of three and one-half weeks' duration, with paracentesis two weeks ago. Prompt operation; sinus and dura exposed; fibrinous exudate over sinus area; spinal fluid pressure 3+, cloudy, 675 cells per c.mm. 50 per cent polys., 50 per cent lymphocytes; no growth obtained. Discharged recovered Aug. 28, 1936.

These three cases, occurring within a few months in one man's experience, are quoted in support of the recommendation for prompt appropriate surgery in all meningitis cases associated with otitis media, in which another cause cannot immediately be established.

As to treatment: relief may be obtained by spinal drainage, whether Kubie's method of forced drainage or by drainage of the cisterna pontis lateralis, combined with forced hypotonic fluids, or by repeated spinal taps, cisternal if necessary. We have recently personally seen two authentic cases of hemolytic streptococcus otogenic meningitis with recovery, and know of others, in which the treatment, in addition to supportive treatment, consisted of the administration of the sulphonate dyes (prontosil, sulfonilamide, etc.).

The recognition of and proper management of petrositis should bring some improvement from the preventive standpoint. I have one case of petrositis with clinical meningitis and high cell count, but with negative culture and recovery.

Methenamine has bacteriostatic value in the spinal fluid (60 gr. per day intravenously or by mouth as long as the urinary tract tolerates it). I know of no cures of meningitis by methenamine after definite onset, but it may have preventive value. Intraspinal injections of diethyl hydrocoprein dihydrochloride, as reported by me some years ago, and colloidal gold, have not succeeded.

For the present our hope lies in recognition of the proper time for preventive surgery; its scope, whether simple, radical, into the petrosa, translabyrinthine, etc., discussion of which would lead into a monograph on mastoid surgery. I emphasize the description "proper time," for I am fully convinced that too early surgical intervention in uncomplicated mastoiditis; *i.e.*, during the congestive state, is productive of much mischief, and that our usual textbook indications lead to this. I do not think there is much danger of waiting too long in a hospitalized and properly observed patient. In our five years or more of service at the children's department of Cook County Hospital, with about an average of 200 population, there have been two deaths from meningitis associated with otitis media that developed after admission. Of these, one was a pneumonia and the meningitis developed in spite of mastoidectomy; in the other, the diagnosis was obscure, and we awaited the result of spinal fluid culture. Since writing the above there has been a case of generalized infection with terminal meningitis which was considered too poor a surgical risk for operation.

What should be our course in the cases with violent onset, which sometimes lead to a fulminating meningitis? My investigation has led to no conclusion as to whether immediate mastoidectomy offers a better hope than conservative treatment and awaiting the immunizing and localizing power of the patient as we do in other infections, *e.g.*, of the face. Perhaps we may take the same course as advocated by Lund in labyrinthine involvement; namely, watch the spinal fluid. In the cases which from the beginning have chills and show the characteristic fever curve of venous infection, blood transfusion or even convalescent scarlet fever serum should be resorted to before exploring the lateral sinus. Many such cases are merely bacteremias with the otitis as an incidental feature. Also other infections must be ruled out, pneumonia, pyelitis, etc.

TABLE I.

SYNOPSIS OF GENERAL RECORD OF CASES.

Case	Age	History	Clinical Diagnosis	TREATMENT		General Autopsy Findings
				Medical	Surgical	
So	3 yrs.	Bilateral otitis. Vomiting.	Meningoencephalitis. Left otitis media.	Continuous spinal drainage.	None.	Diffuse supp. leptomeningitis. Brain negative.
Mi.	3 yrs.	Left otitis, fever opisthotonous.	Left otitis media. Meningitis.	Intravenous-intraspinal conval. serum.	Left mast. dura, both fossae exp.	Supp. menin.
La	6 mos.	Convulsions.	Lobar pneumonia. Meningitis?	Supportive.	None.	Supp. menin. Pneumonia.
Ma	13 mos.	Cough. Convulsions.	Pneumonia; later bilateral otitis, meningitis.	Supportive.	Left mast. dura, both fossae exp.	Supp. menin. Pneumonia.
Se	10 yrs.	Bilat. O.M., headache, fever, stiff neck. R. mast. op. five years ago.	Bilateral otitis, meningitis.	Continuous spinal drg.	None.	Supp. menin.
To	5 yrs.	Scarlet fever, R. otitis, mastoiditis, vomiting, stiff neck.	R. otitis media. Meningitis.	Supportive	R. mast.; dehiscence bone found overlying post. fossa, dura unhealthy.	Supp. menin.
Br	4 yrs.	Bilat. O.M. fever.	Bilat. O.M.; later, meningitis.	Continuous spinal drainage.	R. mast., dura of both fossa exposed.	Basal leptomeningitis.
Cl	5 yrs.	None available. Drowsy; stiff neck.	Epidemic meningitis.	Intravenous & intraspinal antitoxin	None.	Supp. leptomeningitis (influ. bac.).
Ga	5 yrs.	Drowsy; unable to move legs.	Bilat O.M.; meningitis with encephalitis.	Supportive.	L. mast., dura fossae exp.; cisterna pontis lat. drained.	Supp. leptomeningitis.
Al	5 yrs.	T & A recently. Right O.M. Drowsy.	R. O.M. and mast. Meningitis, TB? or suppurative?	Supportive.	R. mastoid, sigmoid sinus healthy.	Supp. leptomenin.; R. petrositis; R. sphen. osteom.
Me	11 yrs.	Left otitis.	Left otitis media. Meningitis.	Supportive.	None.	Supp. menin.
Fr	14 yrs.	Left otitis.	Bilat. O.M. Meningitis? Pneumonia.	Supportive.	None.	Early menin. emboli thru organs. Pneumonia.

TABLE II.
BACTERIOLOGY OF CASES.

Case	SPINAL FLUID Smear	SPINAL FLUID Culture	POSTMORTEM MENINGES Smear	POSTMORTEM MENINGES Culture	FROM EARS, MASTOID During Life	FROM EARS, MASTOID Postmortem
So		Strep. hem.			Strep. (culture)	
Mi	Pneu.	Pneu. III	Pneu.			Pneu. (smear)
La			Pneu.			
Ma			Pneu.			Pneu.
Se	Strep.	Strep. hem.	Strep.			Strep.
To		Strep. hem.	Strep.			Strep.
Br		Strep. non-hem.	Strep.			Strep.
Cl			Influ. bac.	Influ. bac.		Influ. bac.
Ga	Pneu.		Pneu.		Pneu.	Pneu.
Al			Pneu.			
Me	Pneu. III	Pneu. III				
Fr						Mixed infec.

Summary: Strep., 4; Pneu., 6; Influ., 1; mixed, 1.

TABLE III.
RECORD OF HISTOPATHOLOGICAL FINDINGS.

Case	Osteitis	Osteomyelitis Apex	Labyrinthitis Serous	Labyrinthitis Suppurative	Supp. Cav. Tympani
So	R and L	L	L		R and L
Mi	R and L		R and L	R and L	R and L
La[1]			R		
Ma	R and L		R	L	R and L
Se	R and L		L		R and L
To[1]	R		R		R
Br	R and L	L	R and L		R and L
Ga	R and L				R and L
Cl	R and L			R and L	R and L
Al	R	R			R
Me[1]	L			L	L
Fr[1]	R				R

[1]Only one bone examined.

TABLE IV.

DISTRIBUTION OF PNEUMATIC SPACES.

Case	Age	To Canal Region	Beyond Sup. Canal	To Apex	Peritubal
So	3 years	R	L		
Mi	3 years	R and L	R and L	R and L	R and L
La[1]	6 months				
Ma	13 months	R and L			R and L
Se	10 years	R	L		L
To[1]	5 years	R	R	R	
Br	4 years	R	L		
Cl	5 years	R and L	R		R and L
Ga	5 years	R			
Al	5 years	R and L	R and L		
Me[1]	11 years	L			
Fr[1]	14 years	R			

[1]Only one bone examined.

PART II—DR. E. W. HAGENS.

This is a report of the examination of 20 temporal bones from 12 children, age six months to 14 years, who died with meningitis. At entrance all but three cases had a history of otitis media. One child came in with a respiratory infection and later developed symptoms of otitis media and meningitis. Another was admitted with a history of convulsions. The third case was brought in by the police without a history. Meningitis was either suspected or definitely diagnosed before death in all of the 12 cases. A study of the course of each case shows that all but one case had meningitis upon entrance to the hospital. This one child developed her meningitis following pneumonia and otitis media.

A summary of the bacteriology shows that four of the 12 cases were streptococcus (all on culture of the spinal fluid); six were due to the pneumococcus (one on smear, two on culture of the spinal fluid and three on smear from the meninges at postmortem); one case was due to the influenza bacillus (on postmortem smear and culture of the meninges), and one case was a mixed infection (on smear and culture of the ear and mastoid).

As to the treatment of these cases, all received supportive measures, such as salt and glucose intravenously, blood transfusions and serums, including convalescent serum where indicated. In addition, three of the series were put on continuous spinal drainage with subnormal salt solution intravenously. Six of the 12 cases were operated on. At operation the middle and posterior cranial fossae were found healthy in all except one case. In this case a bony dehiscence was found over the posterior fossa and the dura was unhealthy at this point.

At autopsy, 11 of the cases showed suppurative meningitis. In the twelfth case very early congestion of the meninges was found. In one case petrositis and osteomyelitis of the sphenoid was also found.

In general, the gross examination of the petrous bones did not bring out anything more than what is usually seen

in cases with otitis media, or where the mastoid has been operated. In one case, however, the posterior semicircular canal had been cut at operation where the cisterna pontis lateralis was drained.

Microscopic examination of the bones revealed two instances of pneumatization to the apex. One case occurred in a child, age three years, and was bilateral, although more marked on the left side (see Figs. 1 and 2). On this side one cell tract led to the apex above the labyrinth, another below it, and a third between the cochlea and carotid artery. How-

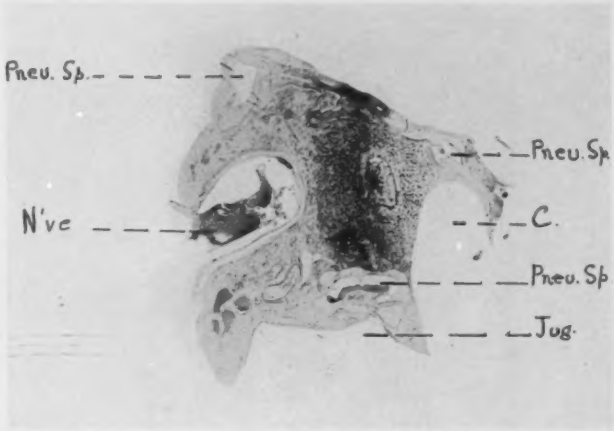

Fig. 1. Vertical coronal section of Mi, age 3 years, left, just anterior to the cochlea. Pneu. Sp. represents pneumatic spaces; N've, the nerve tissue; C, the bony channel for the internal carotid artery; Jug, the space for the bulb of the jugular.

ever, no connection was noted between the infected pneumatic spaces and the meningeal surface of the petrosa. In most of the cases pneumatic spaces were found to extend either just to the semicircular canal region, or for a short distance farther forward from this. In many sections the pneumatic spaces were very close to the posterior or superior border of the petrosa. In some bones the pneumatic spaces extended through the superior semicircular canal arch, or above it, or on either or both sides of the crura. Wherever these pneumatic spaces were found, more or less infection was also noted. The only exception was the one case without otitis

media. On the whole, those spaces nearest the source of infection showed more infection than those farther removed. The tunica propria of the spaces was usually greatly thickened.

Marrow spaces were naturally quite extensive and prominent. Both the red and mixed red and fatty types were found. Marrow was frequently found medial to the labyrinth along the posterior surface of the bone. Infection of marrow (osteomyelitis) in the petrous apex was found on one side of three cases. Two of the three cases had a his-

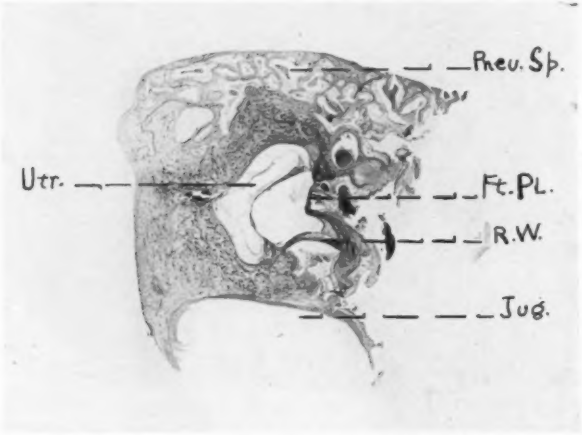

Fig. 2. Vertical coronal section of Mi, age 3 years, left, at level of stapes (Ft. Pl.) and round window (R. W.). Pneu. Sp represents pneumatic spaces; Utr., the utricle, and Jug, the jugular bulb.

tory of bilateral otitis media, while the third case revealed the osteomyelitis on the side having the history of otitis.

Study of the bones showed various interesting connections. The tunica propria of infected pneumatic spaces was seen to connect to the posterior or other petrosal surface in several cases. Evidence of infection in the connections could *not* be shown. Marrow spaces were connected by small channels to a petrosal surface in three bones. In one case polymorphonuclear cells were noted in the channel.

Connections between marrow cavities and the tunica propria of pneumatic spaces were also noted in five of the bones

(see Fig. 3). In one of these the marrow was badly infected, the pneumatic space much less (see Fig. 4). A channel connecting the fossa subarcuata and the tunica propria of a pneumatic space was present in two bones. Infection of the channels could not be shown. Finally, connections were found in two cases between the tunica propria of a pneumatic space or from the mucosa of the cavum tympani to the carotid artery. The presence of pus was noted in one of these connections. However, previous gross examination of the wall of the artery did not show pathology.

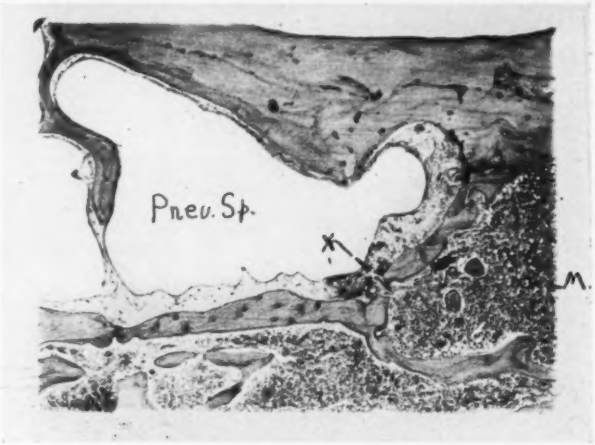

Fig. 3. Horizontal section of Al, age 5 years, left, showing connection (X) between the pneumatic space (Pneu. Sp.) and the marrow (M).

Labyrinthitis was found in all but two cases. In these two cases there had been a unilateral otitis. The labyrinthitis in this series was apparently secondary to the meningitis, for, except in one case, no antemortem connection to the labyrinth from infected pneumatic spaces, marrow spaces or cavum tympani could be found. In this one case some polymorphonuclear cells were seen filtering through a greatly attenuated round window membrane. No perforation was noted, however. Varying amounts of pus were found in the internal auditory meatus in many of the bones. This seemed the most common pathway to the labyrinth from the meninges.

Several of the bones showed diffuse suppurative labyrinthitis. In other cases, only parts of the labyrinth were infected, especially the scala tympani. In several ears the aqueductus cochlearis contained polymorphonuclear cells and in one of these appeared to be the pathway of the infection from the meninges. The pathology in the vestibule and canals was most often in the perilymphatic cavities. Serous labyrinthitis was found in many of the bones, either alone or in conjunction with suppurative labyrinthitis. The case where the posterior semicircular canal was cut at operation died a few

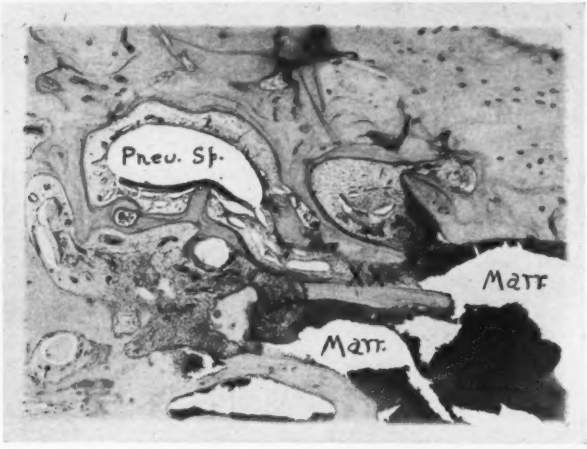

Fig. 4. Horizontal section of Br., age 4 years, left, showing connection (XX) between infected pneumatic space (Pneu. Sp.) and the infected marrow (Marr.).

minutes after operation. The microscopic sections showed partial collapse of the membranous canal; also hemorrhage of the perilymph space here, and hemorrhage in the perilymph in the cochlea, vestibule, and in the ductus and saccus endolymphaticus. In the influenzal case, extravasated blood was found bilaterally in the labyrinth, on one side in the spiral ligament of the cochlea, and the wall of the saccule, while in the other ear blood was noted in the walls of the utricle and saccule. In only one labyrinth could it be said that definite antemortem degeneration of the end-organs was present. It seemed evident that where labyrinthitis was

found it occurred shortly before death, or at least had not been virulent enough to produce marked pathological changes in the end-organs definite enough to be differentiated from postmortem changes. In the one ear showing antemortem pathology the organ of Corti had completely atrophied and the cochlear division and spiral ganglion showed partial degeneration. A study of the child's history showed that a toxic neuritis from measles one year before death was the probable cause of the destruction of the labyrinth.

DISCUSSION.

In the last few years an increasing number of articles have been published on the anatomy and pathology of the petrous bone. Experience has shown that the anatomy may vary extremely. As a result, it is evident that the pathological picture may be just as variable. The potential danger of an infection in the petrosa is thus largely dependent on the anatomic architecture of the pyramid. A petrosa which consists mainly of solid bone with pneumatization limited to the antral region and with small marrow spaces will not likely be a pathway for infection to the meninges. However, where there are extensive pneumatic pathways and/or considerable marrow, infection from the middle ear tract can flood the petrosa and give the opportunity for spread to the meninges.

In the present series, all cases showed considerable marrow. Only two children (one, age three years, and one, age five years) revealed extensive pneumatization to the petrous apex; however, these two cases prove again that such development does occur, although not as often as in adults. Osteitis was found in 11 of the 12 cases. In the twelfth case, no otitis had been present, so that no infection would be expected. Osteomyelitis was also present on one side in three of the 12 cases. Infection probably spreads easily along pneumatic space tracts and, secondarily, along tunica propria connections to marrow spaces, Haversian canals and other channels.

A number of microscopic connections were seen in the various sections. It appears that, anatomically, pneumatic spaces and marrow spaces may be connected with each other, and where either of them are adjacent to a petrosal surface or the fossa subarcuata or the carotid canal, connecting chan-

nels may be present. However, in most of these connections, definite evidence of infection spreading from the petrosa to the meningeal surface was lacking.

It is evident that it is frequently difficult to prove or disprove that infection spreads from the petrosa to the meninges. A gross break to the surface does happen but was not noted in any of the cases in this series. The microscopic connecting channels may or may not have transmitted the infection. Apparently most of the cases developed meningitis in other ways, with an infection of the petrosa also being present. These other avenues of infection have been described frequently in the literature and they afford a probable solution to the meningitis. Extension to the meninges may occur through dehiscence of bone or along vascular channels from the tegmen of the tympanic cavity or antrum. Also, as occurred in one of the present cases, extension may be to the posterior fossa from the sigmoid area. The middle ear and mastoid regions were not furnished to the author, so that examination of these portions was not possible.

Meningitis may occur in cases with ear infection and yet the meningitis need not be secondary to the ear infection. Concomitant sphenoiditis and osteomyelitis has been stressed in the literature as an etiological cause for meningitis. One of the cases in this series had osteomyelitis of the sphenoid as well as petrositis. Meningitis may apparently also occur through a blood stream infection where the origin may be anywhere in the respiratory tract.

From this discussion it appears that in cases of meningitis with ear involvement it is very difficult to have an opinion as to just how the situation has developed. At least, microscopic examination of 20 children's bones (12 cases) does not reveal any constant anatomy or infected pathways through the petrosa.

CONCLUSIONS.

1. The petrous bones of 12 children dying with meningitis have been examined microscopically.

2. The ages varied from six months to 14 years.

3. All but one child had definite otitis media.

4. The bacteriology showed four cases of streptococcus, six of pneumococcus, one of influenza and one mixed infection. The organism was recovered on culture of the spinal fluid in six of the 12 cases.

5. A number of connections between marrow cavities, pneumatic spaces and Haversian canals, and from each of these to the petrous surfaces are no doubt present in all petrous bones; however, they are apparently not a common pathway for extension of infection to the meninges. Infected thrombosed vessels were not seen in this series of bones.

6. In spite of osteitis in 11 of the 12 cases, and osteomyelitis in three cases, practically no evidence of a direct extension of infection to the meninges was found.

7. Labyrinthitis is very common in these cases and in this series was apparently secondary to the meningitis.

8. In eight of the 12 cases pneumatic spaces extended beyond the arch of the superior canal, and in two of these pneumatic spaces were found far forward to the petrous tip.

9. In six of the 12 cases mastoid operation was carried out. In only one of these was extension of the disease to the meninges found, and here the pathology was in the sigmoid sinus region overlying the posterior fossa.

30 North Michigan Avenue.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

SECTION ON OTOLARYNGOLOGY.

Meeting of May 5, 1937.

DR. LAWRENCE R. BOIES showed motion pictures of operations on "Tumors of the Nose and Throat."

DR. FRANK L. BRYANT read a paper entitled, "Anatomical Dissection of Frontoethmoidosphenoidectomy and Operations," giving refinement of technique and showing case exhibits.

An adequate nasofrontal tract is essential in patients on whom an external frontoethmosphenoidectomy is employed. Not all patients present a technical problem. Certain anatomic and pathologic factors may cause an inadequate communication.

The removal of the anterior and middle ethmoid cells and lamina papyracea, with subsequent normal prolapse of orbital tissue, cause an obliteration of the normal nasofrontal tract. Any unusual thickness of the intranasal junction of the nasal and frontal bones and frontal process of the superior maxilla offers the key to the solution. Deep intranasal removal of this thickness offers a practical answer to the problem.